VOLUME II

WILD OATS IN WORLD AGRICULTURE

Edited by: Andrew R. Barr and Richard W. Medd

October 22nd, 1992

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Adelaide, South Australia

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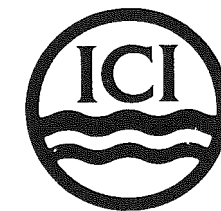Papua New Guinea, or $A50 (plus postage) in all other countries.

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PREFACE

The Fourth International Oat Conference, of which these are the Proceedings, was held in Adelaide from 19th – 23rd October, 1992.

In the business meeting at the close of the Third International Oat Conference held at Lund, Sweden in 1988, the International Organising Committee under the chairmanship of Dr Robert Forsberg was elected to organise the next conference. Adelaide was chosen as the venue. The final date of the Fourth Conference and the outline of the program, including selection of the main speakers, were suggested by the International Committee but local arrangements and finalisation of the program were delegated to a local sub-committee comprising staff from the South Australian Department of Agriculture, the Australian Barley Board, the Uncle Tobys Company and the Australian Oat Breeders Group. I wish to record my appreciation of the work done by this local committee in assuming various organisational responsibilities over the three years prior to the conference.

The Conference was organised into two special symposia ("The Changing Role of Oats in Human and Animal Nutrition", "Wild Oats in World Agriculture"), two days of general sessions (Crop Protection, Molecular Biology, Breeding Methodology, Economics and Marketing, Cytogenetics/Genetics, Physiology, Forage) and a field tour of the Lower North district of South Australia.

The objective of this "Wild Oat in World Agriculture" symposium was to bring together weed scientists, ecologists, oat geneticists and scientists from agrochemical companies to consider approaches to controlling this weed as well as evaluate its role in crop improvement. This assembly of scientists will, I hope, be able to consider the emerging problem of herbicide resistance in wild oats and the related opportunity of breeding for herbicide resistance in cultivated oats.

I would like to thank our generous sponsors. **Incitec** (tea breaks), **Ciba Geigy** (printing), **Du Pont (Australia) Ltd** (printing), **Hoechst Australia Ltd** (assistance for Dr. Devine), **ICI Australia** (assistance for Dr. Morrison) and **Monsanto Australia Ltd** (printing).

The Fourth International Oat Conference also acknowledges the generous assistance for the conference from the following sponsors:

South Australian Department of Agriculture	**Crawford Agricultural Trust**
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Summary

A summative address presented by Dr R. Cousens, School of Crop Science, University of Sydney, NSW 2006, may be published separately to this volume.

The Importance of Wild Oats in World Agriculture

J.H. Combellack
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Summary

The weediness of wild oats is assessed by considering its ecological attributes, occurrence and obnoxious characteristics, and its net economic and intrinsic value. The taxonomy, distribution, reproductive and survival traits and the competitive nature of the species have been asssessed as these form the basis of its negative contributions. The positive qualities of wild oats, such as its value in breeding beneficial traits into cultivated oats and grazing potential have also been explored. It is concluded that too few competition studies exist to enable an accurate assessment of the net negative contributions of this species. Also of concern is the considerable effort placed on modelling when fundamental competition data is limited to a few crops, with few crop/weed densities in a narrow range of geographical locations. It is recommended that more attention be devoted to competition studies to overcome this problem. In view of the considerable research effort, the lack of clear control strategies for farmers is a major worry. It is emphasised that the development of contol strategies that are less dependent on herbicides and which reflect a more accurate assessment of the value of wild oats as a grazing species are needed.

Introduction

Typically plants are designated as weeds by people on the basis of a subjective assessment of their positive and negative characteristics. It can be concluded that the only common features of weeds, including wild oats, is their unwanted occurrence in a given habitat, their undesirable features and their ability to adapt to a disturbed environment[22].

The following formula has been proposed to assess the relative weediness of plants:
(Ecological attributes) x (Land use) x (Net contributions) = (Weediness)[22].
Using this protocol and data contained in substantial reviews[e.g.39,22,28,53] and a selection of the remaining literature this paper will attempt to assess the weediness of wild oats.

Identification

The genus *Avena* (oats) includes both wild and cultivated oats and appears to have originated in the Middle or Near East where early agriculture started[64]. The numerous species of *Avena* have been placed into three categories[68] based on agronomic status viz. (i) cultivated oats, mostly *A. sativa*; (ii) weeds, including *A. fatua* and certain varieties or sub species of *A. sterilis*, the best known being ssp. *ludoviciana*; and (iii) truly wild plants, for example *A. hirtula* (Lag.) and *A. canariensis*.

It has been concluded that a single classification based on phylogenetic relationships was not possible[68] and one based mostly on morphological characteristics, which have been detailed by Combellack[22], is more practical.

Distribution

Wild oats are thought to have originated as a contaminant in wheat grown in Persia and spread mostly by Neolithic Man. More recently this has occurred through the movement of Caucasians[70]. Wild oats are now found from Alaska to Iceland and Iraq to California (USA) in the northern hemisphere and in most non-tropical agricultural areas of the southern hemisphere[37]. *A. fatua* had reached Denmark by the Bronze Age and Britain by the Iron Age[70]. Even so several counties in England were thought to be still free of the weed in 1951

but are now infested. Wild oats can also be transferred in other crops, eg. canola, peas and herbage grasses. Movement of hay and straw has also contributed to their spread, as has farm machinery and straw manure. Birds have been shown to eat large quantities of seed, most of which does not survive digestion[27]. Seeds are also moved by animals and humans.

Avena fatua is the most dominant weedy form in Great Britain, North West Europe and North America. The species is troublesome wherever cereals are grown at 375 to 750 mm annual rainfall[37]. This species has been described as the most widely distributed and troublesome of all the wild oats[37]. Others[70] have suggested that *A. sterilis* ssp. *ludoviciana*, which thrives in Mediterranean climates, is the most abundant and widely distributed. *A. barbata*, though typically a plant of waste places, is found in cereals in N.S.W and Queensland in Australia[70].

When wild oats reaches a new country the most resourceful types are soon selected. This is often reflected as adaptations to day length[70]. While wild oats have colonised virtually every suitable agricultural system, there seems little reason to doubt that it will eventually invade the remaining areas and any others that base their agriculture on temperate crop production.

Survival Features

Seeds of *A. sterilis* ssp. *ludoviciana* survived for up to nine years in England[18] and *A. fatua* for seven years in Canada[5], but in Colorado *A. fatua* was unable to persist for two years[77]. The lack of persistence of the Colorado seeds was suggested to be absence of induced dormancy[77]. Seed banks are exhausted quicker under cropping than pasture though the maximum length of seed survival is similar[18]. Shallow cultivation gives the optimum reduction in seed viability[58,69,75,77] and seed banks therefore decline more rapidly using tyned cultivation rather than ploughing[74] and through spring rather than autumn cultivations[58]. Irrespective of the method of cultivation, substantial increases and decreases in the seed bank have been reported[43,46]. Seed longevity is typically greater at 30 to 34 cm when compared with 0 to 4 or 12 to 16 cm soil depth[18,53,77], but the reverse trend has been reported[18]. There is a substantial loss in viability in the first year after seeding ranging from 55%[74], 74%[5], 77%[77] to 90%[53]. Decline in viability was slower over the following years. Most, 60 to 80%, wild oat seedlings emerge from the top 7.5 cm of the soil although some can emerge from 23cm[38]. Seed survival is longer in lighter and shorter in heavier soils[18]. *A. fatua* seeds have survived 115°C for 15 mins[37]. It has been concluded that wild oat seeds persist for up to nine years and that four to five years is most typical[18].

Seed numbers vary from year to year, with control practices and crop rotations. For example, in a continuous wheat rotation without herbicides, numbers increased from 2030 to 14770 m^{-2} but where herbicides were used the increase ranged from 510 to 6560 m^{-2}[43]. A wheat-sorghum-wheat rotation over the same period reduced seed numbers in the no herbicide treatment from 1950 to 250 and where herbicide was used to a minimum of 50 m^{-2} [43].

Seed Dormancy

Seed dormancy is considered the prime reason for wild oat persistence. Fresh seed is viable and has no dormancy[69]. In *A. fatua* primary dormancy is innate and develops during ripening or later[18]. In *A. sterilis* ssp. *ludoviciana* primary dormancy can be developed while maturing[62]. The proportion of dormant seeds differs between species and strains[58], for example *A. fatua* germinates mainly in the spring while *A. sterilis* ssp. *ludoviciana* is mostly an autumn germinator[18,37]. Seed from the extremities of the panicle are less dormant than those from the centre[18,58]. Also larger panicles tend to have a larger proportion of viable seeds and a lower level of dormancy than from small panicles[37]. The proportion of seeds becoming dormant is also influenced by the temperature during development[65]. Restricting nutrients and water or increasing competition can increase dormancy[37]. While it has been concluded[37] that no generalisations can be made about the dormancy of wild oat species, it has been suggested that three dormancy states exist, two in the primary and one in the secondary dormant caryopsis[1]. Even after considerable research attention the dormancy mechanisms still remain unclear.

Seedling Establishment

A. sterilis (including ssp. *ludoviciana*) germinates and emerges in higher proportions than *A. fatua* at temperatures below 10°C but the opposite is the case above 20°C[17,33,61]. This explains why *A. fatua* mostly germinates in the autumn and spring whereas *A. sterilis* ssp. *ludoviciana* is prominent in the winter[61,69]. *A. sterilis* ssp. *ludoviciana* does not persist in Northern Europe and North America because temperatures are too extreme to allow winter emergence[33]. Soil moisture at the time of emergence is another contributing factor in the distribution of wild oats. *A. sterilis* ssp. *ludoviciana* is troublesome in Mediterranean climates where moisture is often limiting whereas *A. fatua* is mostly found in cool climates with an abundance of moisture[61,70]. While *A. sterilis* ssp. *ludoviciana* appears better adapted to water stress than *A. fatua* one report suggests they are similar[61]. This conclusion is supported by field emergence studies in Spain[4]. Another factor which influences germination is ammonium containing fertilizers. For example, germination and establishment of semi-dormant seeds of *A. fatua* was significantly increased, by 25 to 32%, by limestone, ammonium nitrate and liquid ammonium nitrate in a sandy loam and loam soil[2].

Positive Contributions

Wild oats are used in breeding programmes for cultivated oats. For example wild oat genotypes have been used to impart resistance to pathogens[67,66,36]. Genotypes of wild oats have also been used to impart increased dormancy in cultivated oats in Canada[67,34], dwarfness in Japan[50], assessed for their mycorrhizal infection benefits in the USA[41], for their resistance to aphids in Sweden[72] and appraised for their potential to improve oil and protein content in the UK[71] and India[76] and oil in the USA[13]. Wild oats can also be a useful food source for chickens when included at up to 41% in high-energy broiler diets[9]. *A. barbata* is regarded as an important component of the flora in parts of the U.S.A.[37]. An Indian study has suggested that wild oats have potential value to improve forage oats through a range of traits such as earliness, tallness and tillering[8]. Plants are grazed in pastures, however they do not persist under heavy grazing[55]. Further, a USA study has shown that *A.fatua* had a nutritive quality similar to that of comparable forage species[10]. An Indian study has compared the costs (as economics and energy) of controlling weeds, such as wild oats, in wheat with harvesting them for fodder[3]. It was concluded that harvesting weeds plus grain was as profitable as harvesting only grain[3]. Seeds of wild oat often contaminated other small grain crops and can increase the overall yield of stored grain[37].

Negative Contributions

The most reported negative aspect of wild oats is its competitiveness with crops. This effect in turn has been related to its economic impact on cropping. These will be considered separately.

Competitiveness

(a)   **Barley.** A USA competition study reported grain yield losses of 40% with a wild oat density of 170 plants m^{-2}[52]. In another USA study where wild oat density varied from 60 to 180 and barley by 135 to 415 plants m^{-2} grain yield was reduced from 8.9 to 47.7%[31]. The same authors reported on another trial in which wild oat density varied from 195 to 590 and barley density from 170 to 625 plants m^{-2}. In this trial grain yield reduction varied from 25.5 to 77.5%[31]. In one North Dakota study 84 plants m^{-2} reduced grain yield by 7%[7] and another 100 plants m^{-2} reduced yield by 15%[53]. However, in a dry year (two-thirds of normal rainfall) 11 wild oat plants m^{-2} reduced spring barley yields by 18% in the USA[52]. In France 48 plants m^{-2} reduced yields by 18%[35]. In one English study 218 and 306 stems m^{-2} at harvest reduced yields by 26 and 32% respectively[20] and in another 54, 46 and 15 plants m^{-2} imparted 16% and two non significant yield losses[57]. Canadian studies[28] indicated that barley yield losses are typically relatively insignificant (<10%) though variable at wild oat densities less than 100 plants m^{-2} under normal rainfall conditions.

Wild oat competition was found not to begin until the four leaf growth stage of spring barley in England[20]. In a further trial in England crop and weed density and nitrogen fertilisation affected crop yield[57]. At the highest crop and weed density, 416 and 414 m^{-2} respectively, the critical time of removal was 2.5 to 4.5 leaves but at a crop and weed density of 464 and 336 plants m^{-2}, and where N was applied, no losses occured unless the weed remained until after the 6 leaf stage. The critical period of wild oat interference in spring barley in USA was reported to be between the two-node and heading stages of wild oat at densities of 170 m^{-2} in an Idaho trial[52,51]. In the latter trial, yield was not reduced if wild oats were removed at or before the two-node growth stage of the crop but removal at heading resulted in a 23%, and where not removed, a 40% crop yield reduction.

(b)   **Wheat**.   It has been suggested that wheat is equally as susceptible to wild oat competition as barley[21]. Several North American authors have shown that wheat yield decreases as wild oat numbers increase[20,7,14,12,15]. In North Dakota studies 84 wild oats m^{-2} reduced yields by 22%[7] and 100 m^{-2} by 30%[53]. In a Canadian trial 84 plants m^{-2} were necessary to signicantly reduce yields, in non fertilised plots, and then in only one of the two years of the study, but where fertilized the mean reduction over the two years was 31%[12]. An Australian study in which weed numbers per m^2 varied from 25 to 300 and the crop by 25 to 200 m^{-2} showed that both crop and weed density were important in determining wheat yield reductions[44]. At the lowest wheat densities and the highest wild oat densities in two trials grain yields were reduced by 78% in one and 77% in the other. At the highest wheat sowing rate and highest wild oat densities wheat yield was reduced by 64 and 44% in the two experiments[44]. In another Australian trial[63] wheat density varied from 25 to 300 plants m^2 and wild oats from 8 to 61 m^{-2} in one year and 25 to 188 m^{-2} in another. Wheat yield was reduced by an estimated 50% in one year and by 60% in the other at the highest wild oat and lowest wheat density. However, where there was a high wheat and wild oat density grain yield reductions were 8 and 25%, respectively. These results suggest that increasing crop density reduces the impact of wild oat competition. In yet another Australian study weed density was varied from 0,25,50,75 to 100% of the original population by thinning and superimposed with three nitrogen levels[73]. The data was subjected to regression analysis and averaged over the two years encountered. It shows that at 50 plants m^{-2} yields would be reduced by 450 kg ha^{-1} and at 100 m^{-2} by 600 kg ha^{-1}. A Californian study[14] also found that yield loss related to both crop and weed density, and that at 300 and 100 plants m^{-2}, for wild oats and crop respectively, the yield reduction of the crop was 70% but at 300 and 700 plants m^{-2} the yield reduction was approximately 55%. The yield reductions in these trials[14] suggest that wild oats was more competitive than in other North American experiments[7,12]. They are however similar to a previous Californian study[15] where at approximately 50 plants m^{-2} yield reduction varied from 35% at low crop density to 65% at high crop density. This trial also showed that drilled wheat was more susceptible to competition than broadcast. It has been inferred that wild oats produce variable competitiveness reflecting differences between sites, wild oat biotypes and or crop competition[14].

Other factors involved in determining the effects of wild oats include the critical period of competition. It has been suggested that in Canada wild oat competition in wheat might occur before the crop has emerged[12] or at least before the 2 to 3 leaf stage[21]. In England, studies in spring wheat indicate that competition commences at the four and a half leaf stage[20]. Winter cereals in England are reported to be more competitve with spring germinated *A. fatua* than spring cereals[21]. A New Zealand greenhouse trial showed that when *A. fatua* and wheat were planted simultaneously *A. fatua* was the more competitive due to greater root competitive ability[42]. However, when *A. fatua* was sown 3 to 6 weeks later than wheat, the wheat was the more competitive. Wheat grain yield in wild oat infested plots typically declined with fertilisation while the density of wild oat panicles increased[14]. It was concluded that one would be ill-advised to apply nitrogen to a wheat crop unless wild oat numbers were low or could be controlled.

(c)   **Flax**.   Limited competition studies indicate that flax is less competitive than wheat. For example on summer-fallow in Canada 48 plants of wild oats m^{-2} gave a mean reduction of 41% over three years and on stubble 60%[12]. In a USA trial 48 m^{-2} reduced yield by 57% with and 53% without fertiliser while the highest density of 192 m^{-2} reduced yields by 86% with and 83% without fertiliser[7]. In another USA report 100 wild oat seedlings m^{-2} reduced flax yields by 65%[53].

(d)   **Lentils**.   It has been shown that wild oat infestations of 32 and 65 plants m^{-2} do not affect yield if not removed for up to five weeks. However, if the same infestations were not removed for 7 weeks then a 32 and 42 % reduction was found and after 11 weeks it was 49 and 61%[25].

(e)   **Other crops**.   The following have reportedly suffered yield loss due to wild oat competition; sugarbeet[32], peas[21], sunflower[30] and maize[16].

These data clearly show that wild oats are competitive with a wide range of mostly temperate crops. However, the density of wild oats necessary to reduce yield varies from crop to crop, the duration of the infestation, the vigour of the crop and among geographical locations. The reviewed competition studies imply that wild oats are typically less competitive than sometimes inferred by authors who base their assumptions on herbicide trials. Additionally, in view of the supposed importance of wild oats as a weed, relatively few studies were found that critically assessed their relative competitiveness. Also of concern are the numerous models developed around such limited data. It is suggested that a series of fundamental experiments need to be conducted, in at least wheat and barley, to more accurately delineate the critical time of competition and the relative competitiveness of wild oats with crops in the major growing areas of these crops. Such data is essential if one is to generalise on the relationship between crop yield depression and wild oat density[22].

Impact

It has been suggested that the competitive effect of wild oats reduces the annual production of wheat and barley by approximately 13 million tonnes, sufficient to feed 50 million people at subsistence level[29]. Some 11.3 million ha of wheat and barley in the USA were estimated to be infested with wild oats[29]. In 1976 the losses due to wild oats in the USA were put at $150 to 200 million[29] and in Canada at around $200 million in 1976[29]. The annual cost of wild oats to the Australian wheat industry was estimated to be $42 million in 1990[47].

Much of the economic data on wild oat control has been generated following the use of herbicides. A USA (North Dakota) experiment showed that crop yield with no weed control treatment was 599 kg ha^{-1} but where triallate + bromoxynil + diclofop was used it was 1426 kg ha^{-1} and a net return of $113 was obtained[54]. As density of wild oats, and the range and density of other species are not given, these data are of limited value. A New Zealand study showed that the break-even yield loss varied from 6.4 to 41.1% for wheat and 9.1 to 20.2% for barley based on a crop yield of 4.5 t ha^{-1}[11]. In Canada in continuous wheat or in a barley/canola rotation *A. fatua* control every year provided the best economic return but in continuous barley control every 2 or 3 years was optimal[56]. An English model predicted that the highest long-term benefits in wheat would be obtained if a herbicide was used every time the density exceeded 2 to 3 seedlings m^{-2}[23]. A USA study on barley has shown that at a wild oat density of 100 plants m^{-2} net return was greatest when half the recommended dose rate of the herbicide was used but at 290 m^{-2} it was greatest when half or full dose rates were applied[6].

In summary, there is only limited economic data for such an "important" weed. Further, the available data demonstrates that it is extemely difficult to predict economic returns from control strategies particularly when there is limited competition data and the weed exerts a variable influence on crop yield depending upon crop/weed density, soil type, fertiliser and geographic location. Also, as recently pointed out[54], "farmers presently feel that their own, or their consultants judgement, is sufficiently precise and less demanding on their time than available models". Making more informed decisions in the future will be dependent upon the generation of user friendly models based on sound competition studies. In short, more attention needs to be placed on data generation than data manipulation.

Yield reduction is not the only deleterious effect of wild oat competition. While in wheat substantial yield reductions are generally not accompanied by an adverse effect on protein content[12], in flax, oil content has been greatly reduced even with low densities of wild oats[21]. An English study has reported that the percentage of harvested grain contaminated with wild oat seeds increases as its density in the crop increases[24]. The study also showed that barley was more prone to contamination than wheat. Only 2 out of 950

silos recorded seed contamination levels above the threshold of 50 wild oat seeds per 0.5 litres in an Australian study[48].

Management and Control

The negative effects of wild oats mostly pertain to crop production. Since different control strategies affect the level of control, farmers must be knowledgeable of the differences when setting their objectives. To many, the principal objective is to achieve the best economic return with secondary objectives being visual appearance and long term farming with cropping options not being prejudiced[26].

Herbicide use has been the dominant control method for over three decades. The range of herbicides marketed enables effective control in most crops. The more widely used herbicide groups are: amino proprionic acids; carbamates; ureas; triazines; sulfonyl ureas; imidazolinones; dinitroanilines; aryloxyphenoxypropionates and cyclohexanediones. As wild oats have confirmed resistance to a number of herbicides[59,40] reliance on herbicides acting on the same metabolic target site must be avoided. To ensure long term herbicide potency farmers must adopt strategies which prevent herbicide resistance[45,59]. This can be achieved through crop rotation, ensuring alternating herbicides which have different modes of action and by minimizing seed production. Recent Australian data indicates that seed set is minimized if the wild oats are sprayed between stem elongation and booting[48,49].

Conclusions

Even with all the research effort, it is regrettable that a concise systems approach to wild oat control has not been spelled out. Some of the obvious tactics are:

i) when harvesting, collect and burn all seed. This will of course be of limited value in some crops as wild oat tends to shed its seed before many harvest;

ii) adopt a rotation that is known to suppress wild oat survival, for example, wheat/sorghum/wheat;

iii) reduce seed production by selecting the most competitive crop or crop variety, planting the crop at a higher than normal density and/or by reducing seed set with selective herbicides;

iv) prevent seed burial by minimising soil disturbance; and

v) ensure a high level of seedling mortality through shallow cultivation and or herbicides.

From the available data, implementing such strategies should reduce wild oats to very low levels within four to five years in a continuous cropping system. Farmers confronted with the task of appraising the value of wild oat control in the crop need a simple user friendly model to enable more accurate predictions of risks and benefits. Most models necessitate an estimate of weed free yield at the time of treatment, an almost impossible task. In one study it was shown that the competition index for a weed is apparently independent of crop yield and it was suggested that a simple model based on a linear regression coefficient can be used in a predictive way[73]. Alternative approaches are (1) to estimate the weed free yield and then utilize regressions calculated using square root transformations of wild oat density[28] or (2) by fitting the density/competition information data to a rectangular hyperbola and incorporate expected crop price and control costs in the model to determine expected profits/losses[67].

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Biological and Agronomic Reasons for the Continuing Importance of Wild Oats in the United Kingdom

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Summary

The changing status of wild oats in the UK over the past 20 years is reviewed in relation to their biology and agronomy, to the availability of effective herbicides and to increasing pressures on farmers to reduce herbicide use. Early emerging wild oats can be particularly competitive in autumn sown cereals when followed by mild winters. Average grain yield losses of 1% per wild oat plant per m^2 have been used for defining loss thresholds; such losses have been shown to vary with the density of the crop and with the level of applied nitrogen fertiliser. The rate of seedbank decline is influenced by seed age and by cultivation. Strategies are discussed which integrate cultural measures and herbicide use with the aim of depleting the seedbank in the long term. Forecasting wild oat emergence from the seedbank is difficult because seed dormancy varies with phenotype, with the environmental conditions under which the parent plant is grown and with the position of the seed within the spikelet. Economic pressures on the farmer resulting in less expenditure on herbicides, together with more 'setaside' land taken out of production and a ban on straw burning, are factors which are likely to result in an increase in wild oat infestation in the future.

Introduction

Cereal cropping systems in the United Kingdom are based largely on wheat and barley, interspersed with break crops such as canola and legumes. Cultivated oats are grown over a relatively small area. Most of the wheat, about half of the barley, and nearly all of the canola and bean crops are sown in the autumn. A survey of herbicide use on farms in England and Wales in 1990[8], showed that grass weed herbicides were used more than once on many crops of wheat and barley to control the commonly occurring annual grass weeds — wild oats (*Avena fatua* and *A. sterilis* ssp.*ludoviciana*), black grass (*Alopecurus myosuroides*), brome species (*Bromus sterilis, B. commutatus, B. hordeaceous* and other species) and the meadow grasses (*Poa trivialis* and *P. annua*). Herbicides aimed more specifically at the control of wild oats were used on about three quarters of the wheat and barley. In contrast, only a quarter of the oat crop was sprayed for grass weeds, mostly meadow grasses, and no herbicides were available for wild oat control. The difficulty of controlling grass weeds in oats is an important reason for the lack of expansion of this crop when compared with wheat and barley in the United Kingdom.

Cereal yields have increased over the past 20 years with the introduction of new varieties. High yields, sometimes over 10 t/ha of winter wheat, are associated with the long growing season (300 to 330 days from sowing to harvest), and with adequate moisture available during the growth period. In autumn sown crops, wild oats which emerge at the same time as the crop or before the crop has 2 to 3 leaves (usually early October to early November), can be very competitive. *A. fatua* is the most commonly occurring species[1], often misnamed the spring wild oat, and it can germinate over a six month period between October and April. The winter-germinating *A. sterilis* ssp.*ludoviciana* also occurs but less frequently than *A. fatua*.

Surveys of wild oats in the 1970s and early 1980s[1,4,5] showed a change in the pattern of infestations. High densities of wild oats had been common in cereal crops until the mid 1970s, with wild oats being the most serious annual grass weed in the UK. The introduction of new, and more effective herbicides, coupled with a big increase in cereal prices in 1974, meant that from the mid-1970s farmers had effective and affordable wild oat herbicides. Some farmers, mostly seed growers, embarked upon an eradication policy for *A. fatua* by

combining herbicide use with the hand roguing of any survivors. However, most farmers adopted a strategy of containment, a low- cost approach for living with low levels of wild oat infestation. This has led to the present situation where wild oats are widespread at low infestations, and pose a threat of build up if control measures are relaxed. Wild oats are now regarded as being of less importance to the cereal farmer than are brome species and black grass, weeds with a higher potential rate of increase relative to herbicide performance. This was shown in a recent farm weed survey when grass weed herbicides, used annually, reduced wild oat populations to very low levels, but those of black grass remained relatively constant over a period of ten years[19,25].

Pressures on farmers to reduce the use of herbicides are increasing, and it is often difficult to decide whether the cost of herbicides is justified in efforts to control low wild oat populations. The farmer may opt for lower rates of herbicide, accepting the greater risk of poor control. A lower dose applied to a lower threshold population may be more economic in the long term than using the recommended rate at a higher threshold population. To devise economic control strategies, farmers need to know how crop yield and wild oat seed return might respond to reducing herbicide dose, together with information on the competitiveness and the spatial and temporal dynamics of wild oat populations.

Biology and Agronomy

Wild oat has been a key species for experimentation to establish some of the principles of weed competition. In a comparative study between weed species at IACR Long Ashton[26], wild oats were the most competitive of 12 autumn-germinating weeds in winter wheat. In this experiment, wild oats grew without the usual winter check to growth following a very mild winter, and were exceptionally competitive, reducing yields more than cleavers (*Galium aparine*) which is usually our most competitive weed.

A rectangular hyperbolic relationship[2], fitted to our data sets, suggests that low wild oat densities are likely to result in yield losses in cereals of 1% for every wild oat plant present per m^2. Such a figure has been used to predict yield loss from low density infestations, and to define economic threshold populations where the value of the yield loss from wild oats is less than the cost of control. At current grain and herbicide prices in the United Kingdom, the yield loss from 1 to 2 plants per m2 is unlikely to warrant the cost of spraying. But farmers' pride in having fields free from conspicuous weeds, and their concern over allowing weeds to seed and risk building up future infestations, are strong reasons for controlling low density wild oat populations.

Simple models of crop losses due to wild oats are useful for description and generalised forecasting, but have limited value for predicting the effects of specific infestations of wild oats. Many factors can affect the outcome of competition, and studies of the effects of such factors as crop density, nitrogen fertiliser and relative time of emergence, aim to describe some of the variability in competitive effects from wild oats.

Larger competitive effects, at a low crop density, were shown in a study[21] where a wheat yield loss of 1.2% per wild oat plant per m^2 was recorded in a crop of 134 wheat plants per m^2. This compared with a 0.7% loss per wild oat in a crop of 443 plants per m^2. Thus, lower threshold densities are needed for thinly established crops.

Also, nitrogen fertiliser has been shown to favour wild oats at the expense of wheat[27]. Wheat yields in the absence of wild oats increased from 4.3 t/ha to 6.7 t/ha when nitrogen applications were increased from 0 to 200 kg/ha. Yields in the presence of 45 wild oat plants per m^2 were 2.1 t/ha (51% yield loss) and 0.7 t/ha (90% yield loss) when nitrogen was increased from 0 to 200 kg/ha.

In autumn sown crops, wild oats which emerge with the crop before early November are more likely to become well established and survive the winter than those emerging later. Their chance of survival increased markedly as soon as the second leaf appeared[6], and plants which had reached three leaves by mid-December suffered only minor mortality (<20%) compared with plants with only one leaf at this time (>70% winter mortality). Greater competition and seed production resulted from autumn emerging wild oats compared with those emerging in the winter and following spring[23]. Even where more seedlings emerged

in the spring than the autumn, these were severely suppressed by crop competition and total seed production was nearly all derived from the autumn fraction of the population.

To reduce wild oat populations the farmer needs a strategy to deplete the seed bank, by preventing the addition of new seeds, and by encouraging losses of existing seeds from the seed bank. This calls for an integrated approach which combines herbicide use with cultural measures and for an understanding of the factors governing seed loss. Studies of the population dynamics of wild oats have been made with the aim of forecasting changes in wild oat populations resulting from different management systems[20].

In arable crops wild oats appear to be less persistent in the soil than many broadleaved weed species[13]. We have recorded, in the absence of further seeding, typical rates of seed bank decline of 50% of new seeds in the first year and 90% per annum from older seeds in subsequent years[17]. Losses of dormancy as seeds aged resulted in proportionally more seedlings emerging from the older seed bank in the second and subsequent years than from new seeds. Therefore, with complete wild oat control, the annual seedling population will decline more slowly than the seed bank. Thus, effective herbicide control needs to be maintained for at least three years to reduce high infestations to low levels.

Cultivation is recognised as a stimulus to wild oat germination[16], and is a key factor controlling the persistence of wild oats. In a six year experiment[24], three years of cultivation for spring barley exhausted the seed bank when no seeding was allowed. In contrast, with no soil disturbance, seeds persisted under grass for six years, allowing wild oats to appear in the following wheat crop. On undisturbed direct drilled soil a small proportion of the original seeds persisted after four years[18]. It seems likely that a small proportion of a natural population could persist for many years in soil which remains undisturbed.

Much work has shown that the type of cultivation has a strong influence on populations of short dormancy weeds[3]. Shallow non-inversion tillage retains seeds in the surface soil and encourages wild oat populations to increase when compared with ploughing. For example, seedling populations increased by factors of 3.5 x after tyne cultivation and by 1.7 x after ploughing with no herbicide control in successive spring barley crops[22]. Increased germination after shallow tillage means that with good control, the seed bank will be depleted more quickly than after ploughing. With poor control allowing seeds to shed and return to the soil, there will be less prospect of reducing the seed reserve with shallow tillage than with ploughing. Thus, strategies employing reduced rates or cheaper, less effective, herbicides with a higher risk of poor control, should be allied to a ploughing rather than to a shallow tillage regime.

A wild oat population model based on experimental data[20], shows the level of control needed annually to prevent the population increasing. This varies between 70% (ploughing and burning straw) and 85% (tyne cultivation without burning). Weed species like black grass, with a higher potential for increase than wild oats, need a higher level of kill (up to 97% for non-ploughing systems) for containment[9]. This is not easy to achieve consistently, and makes black grass currently a more serious weed problem than wild oats in the United Kingdom.

The wild oat model predicts average rates of population change which agree broadly with those found in practice[14]. Selman reported that, over five years, uncontrolled wild oat populations increased by an average factor of 2.7, with annual increase factors which varied from 1.3 to 6. Decrease factors with herbicide use showed similar annual variation. These annual fluctuations were related to delayed sowing, poor seedbeds, and variation in time of harvest, crop competition and herbicide efficiency. Such a model based on average factors is useful for describing long-term population trends, but its value in forecasting the numbers of wild oats likely to occur in the next year is limited, because of variability in those factors that govern population change.

Different phenotypes of *A. fatua* show variation in seed dormancy. The colour phenotypes fA (brown seeds) and fB (grey seeds)[15] showed differences in emergence behaviour[11]. Seeds were collected from six sites; there was considerable variation in dormancy between sites within a given phenotype. Within sites there was also variation between phenotypes; at five sites fA was less dormant than fB, but fA seeds from the remaining site were more dormant than fB. Variation in seed survival in the soil was also found. Collections from the same six sites gave an overall survival of viable seeds of 1% after five years. However one

phenotype (fA) from one collection showed a survival of 23% of viable seeds after five years[11].

The environmental conditions under which the parent plant is grown, and the position of the seed in the spikelet, also affect seed dormancy. In growth chamber studies, hot or dry environments or a combination of both can result in plants producing less dormant seed. Seeds from plants grown at 20°C with or without water stress gave 78% and 30% emergence, and seeds from plants grown at 15°C gave 47% and 10% emergence with or without water stress, respectively, in the autumn after shedding[10]. Basal seeds in spikelets are less dormant than distal seeds, so that germination in the first autumn immediately after shedding is mostly from basal seeds. The greatest emergence from shed seed occurs in the second spring due to the contribution of distal seeds which by this time have started to lose their dormancy[12]. Variation between years in the amount of chilling can also give rise to differences in numbers emerging. Flushes of emergence occurred in warm periods immediately following periods of chilling below 4 to 6°C[11,12].

Wild Oats in the Future

The availability of effective herbicides means that farmers, at present, are less concerned about wild oats in cereal systems than they are about brome species and black grass. Even though cereal prices in real terms have been gradually declining since the early 1980s, farmers have been able to afford and achieve effective control and containment of wild oats.

At the time of writing, future prospects for cereal growing are uncertain. The recent review of the EC Common Agriculture Policy means a price reduction for grain of about 15%, or £15 per tonne, to bring it nearer to the world price, together with 15% of arable land to be taken out of production ('setaside'). It is unlikely that the agrochemical industry, faced with a severe diminution in volume of sales, will concede a corresponding reduction in herbicide prices to the farmer to maintain the existing cost/benefit ratio. As a result farmers will be able to spend less on herbicides and have to rely increasingly on integrated strategies of low doses of herbicides allied with cultural measures. Alternatively, this might provide an incentive for a chemical company to lower the price of their wild oat herbicide to the farmer, possibly by reducing the technical back-up and field support of their product.

Declining cereal profitability may mean that fewer cereals and more break crops such as legume crops and linseed are sown. Break crops which are sown in the spring should diminish the problem of black grass and brome where germination is mainly confined to the autumn. Spring-germinating wild oats could be a problem in crops such as spring sown beans, which tend to be poorly competitive, and could allow the recovery of wild oat plants which survive herbicide use.

Declining profitability may also mean that farmers who, at present, use an autumn prophylactic spray followed, in the majority of cases, by a spring herbicide, may have to consider cutting the cost of the autumn prophylaxis by perhaps resorting to triallate (£23/ha), targeted for wild oats but giving some control of other grasses and broadleaved weeds, instead of using triallate plus isoproturon (£50 to £60/ha) which gives a broader spectrum of control. If further wild oats emerge in the spring, this cheaper option could be followed by a spring herbicide, with the possibility of adjusting the dose according to growth stage and environmental conditions at the time of application[7]. Such a possible strategy can only be considered here in terms of general principles rather than for specific field situations.

At present it is not clear how the increased area of 'setaside' land is to be managed, but there is a real risk of wild oats building up if this land remains uncropped. Straw burning destroys a proportion of newly shed seeds[22], so the 1993 ban on straw burning is likely to exacerbate the wild oat problem. We feel that over cereal systems as a whole, there is a real danger of wild oats increasing because of 'setaside', the ban on straw burning, and the prospect of greater economic pressures on the cereal farmer.

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Understanding Seed Dormancy in Wild Oats (Avena fatua) and its Implications for Control Strategies

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Summary

Seed dormancy is a genetically controlled lack of synchronism in the development and functioning of the structural and biochemical components of the seed. The environment can induce this asynchronism during seed development on the parent plant and after seed abscission. Light, temperature, gaseous composition, inorganic and organic substances and particularly water status may act individually, or interact, to induce, sustain and terminate dormancy in different ways according to genotype and age. Because of genetic heterogeneity for seed dormancy in natural populations, control of dormancy will be difficult. Weed control measures should aim at reduction of fecundity to reduce seed-bank size.

Introduction

Seed dormancy is considered to be a polymorphic character in both wild oat (*Avena fatua*) and *Avena barbata*[14]. Self-pollination is predominant but outcrossing can range from one to 12 per cent[13] so that even in local habitats considerable diversity in genotype can occur. Genotypes range from those expressing complete absence of dormancy at the time of abscission from the parent plant to those showing persistent dormancy for as long as seven years[24]. Within hexaploid oat species *A. sativa* has the shortest and *A.fatua* the longest period of dormancy. Any explanation of the nature of seed dormancy in the wild oat must take account of this genetically determined diversity that can be modified in many different ways by the surrounding environment during the development of the seed on the parent plant, and after the seed has been separated from the parent by abscission. The objective of this paper is to demonstrate some of the complexity of the interactions between genotype and environment that create the great range of expressions of dormancy in the environmental landscape. It is because of this diversity of expression and the unpredictability of its occurrence that control measures aimed at eliminating dormancy are not likely to be successful in the field. Measures that reduce fecundity so that the soil bank of dormant seeds is minimized should be the first approach in the practical control of the wild oat as a weed in cereal crops.

The Nature of Seed Dormancy in the Wild Oat

The role of the parent plant and seed structure

The position of the caryopsis on the parent plant, the physical nature of the inflorescence with its lemma, palea, glumes and awns together with the specific nature of the pericarp and testa (seed coat) all contribute to variation in the expression of degree of dormancy. These positional and structural influences are particularly important during the development and maturation of the seed on the parent plant because they determine the degree of primary dormancy found in the floret when it separates from the parent plant[34]. Secondary and tertiary florets are more dormant than primary florets and are shed earlier from the inflorescence[27,28]. Florets at the bottom of a panicle are more dormant than those at the top[31]. Genotypes with long grain development times are generally more dormant than those with short development times[1,2]. The lemma and palea (commonly called hulls) that surround the caryopsis (fruit) have important effects on the expression of dormancy because they appear to have chemicals that inhibit germination, and can limit gas exchange and water uptake[34]. Dehulling generally promotes germination and speeds up the natural loss of dormancy through the acceleration of after-ripening[34].

The most important physical structure that influences dormancy is the seed (caryopsis) coat, comprised of the outer pericarp and inner testa. The testa has in addition an inner and outer cuticle that surrounds the entire caryopsis except in the area adjacent to the embryo[7]. There have been at least 25 reports that breaking the seed coat, for example by pricking, and dry or chemical scarification, can break a high proportion of the dormancy expressed in a population of seeds[34]. For a long time it was thought that pricking the seed enhanced availability of oxygen but more recent evidence suggests that a more important effect is the enhancement of the entry of water[22]. Pricking the seed close to the embryo induces a higher germination than pricking at the distal end of the caryopsis[29]. Coat-imposed dormancy is common in grasses[34] and many of the seed-environment interactions leading to variation in the expression of dormancy are mediated through changes in the permeability of the seed coat to water and gases.

Removal of hulls and puncturing the seed coat does not eliminate dormancy in all genotypes of the wild oat. Genotypes with very persistent dormancy have a form of embryo dormancy that persists even when the embryo is excised from its surrounding structures[34]. The mechanism of this embryonic dormancy is very complex and distinct from that imposed on the embryo by the covering structures. Protein synthesis, carbohydrate metabolism and plant growth regulators are significant in the expression of embryo dormancy. Key environmental factors such as temperature, light, and water play important roles in controlling the induction, maintenance and termination of embryo dormancy. Genotypes that express prolonged and deep dormancy generally have both embryonic dormancy and seed coat-imposed dormancy. Primary dormancy is lost over time by the natural processes of after-ripening and embryonic dormancy is generally lost before the dormancy due to covering structures is terminated. Alternatively embryo dormancy may be re-introduced after it has been terminated (secondary dormancy) through interactions between the environment and the covering structures of the caryopsis.

Most modern cereals do not express seed dormancy as an embryonic dormancy indicating that this character has been selectively removed by the selection for uniform crop germination[34]. This is not to say that other forms of dormancy induced by the environment are not expressed through failure of the embryo to germinate. The test for embryonic dormancy is to excise the embryo completely from its surrounding structures and germinate on water at the optimal temperature. It is clear that in embryonic dormancy a number of metabolic blocks can be present that are sequentially lost during after-ripening[3]. Genotypically distinct pure lines have been isolated reflecting a range of expressions of dormancy from the complete absence of dormancy to deep dormancy reflecting the expression of a number of metabolic blocks present together in a single genotype[24,36]. At least three genes have been indicated in the expression of dormancy in the wild oat with a strong influence of the maternal tissues[15] on embryo germination and at least one gene affecting metabolic activity of the embryo[17].

Environmental influences on seed dormancy

Superimposed on the above expressions of dormancy that are inherently related to the genotype, physical structure and maternal-zygote relationships of the wild oat, are the effects of environmental changes throughout the life cycle on both parent and offspring. Examples of the key factors of water, radiation (heat-cold-light-photoperiod) gases, growth regulators and agricultural chemicals will be used to illustrate how they interact to provide the plasticity in expression of dormancy.

Water. Water stress on the parent plant during maturation can reduce the level of seed dormancy[21]. Alternate wetting and drying decreases germination by inducing secondary dormancy[18] and in wet soil or in high humidities on the soil surface seeds can be induced into secondary dormancy[32]. On the other hand a moist atmosphere combined with high temperature can hasten the loss of dormancy in stored seed[26] but under similar conditions in wet soil the state of dormancy can be sustained for as long as seven years[33]. Embryos seem to have an absolute requirement for exposure to a liquid phase of water to be able to terminate dormancy and this seems related to the inability of the embryo to develop a sufficiently negative water potential to extract water from the surrounding tissues[4].

Radiation. Energy from the sun influences seed germination in two important ways. Wavelengths longer than 3000 nm (heat) and between 380-800 nm (light) have separate

but often interacting effects on seed germination and dormancy. Buried seeds are mainly influenced by temperature and moisture changes whereas seeds on the soil surface can be directly influenced by marked diurnal and seasonal changes in photoperiod, light intensity and quality, and relative humidity[34].

Temperature.   There are four ways that temperatures influence seed germination in the wild oat:

1. Induction of seed dormancy during development on the parent plant is strongly influenced by temperature: Low temperatures during seed development enhance dormancy of the mature seed and high temperatures diminish dormancy[34].

2. Persistence of the primary state of dormancy in a mature seed is affected by temperature: Low temperatures favour loss of dormancy and high temperatures, particularly if associated with high humidity, favour persistence of primary dormancy[34].

3. Reinforcement of the primary state of dormancy, or induction of a secondary state of dormancy, may occur in response to temperature changes at any stage of the post-abscission period of the seed in those genotypes that express primary dormancy[34], and

4. After dormancy is lost temperature is a major determinant in the triggering of germination.

The influence of temperature during seed development on the parent plant, on the subsequent level of dormancy found in the mature seed, provides an excellent illustration of the plasticity of response to temperature available within a single genotype. This plasticity shows that arbitrarily dividing phenotypically distinct lines into genetically distinct lines on the basis of their response to a single germination temperature can lead to a lot of confusion about the basis of temperature effects on germination. When the progeny of an apparently dormant group of distinct lines was germinated over a range of temperatures between 4 to 32°C. some of the lines germinated at all temperatures. In others there were varying degrees of germination suppression particularly over the mid-range temperatures. When the plants from the apparently non-dormant lines were grown at three different day/night temperature regimes (28/22,20/20,15/10°C) the progeny showed quite different responses at either 4, 20 or 28°C. As the temperature was increasingly lowered during development there was a progressive increase in dormancy of the mature seeds when germinated at high temperatures (above 20°C). In this way low temperatures experienced during seed development on the parent plant induced a sensitivity to high temperatures at the time of germination. Thus exposure of maternal tissue to a specific temperature creates a particular response to temperature in the progeny in the subsequent growth season[23]. The classification therefore of apparently dormant or non-dormant lines reflects the conditions of seed development and maturation and the germination response at some specified temperature. To describe a seed as dormant, or non-dormant, thus requires a specification of the germination temperature.

Genotypes classified as dormant can after-ripen at different rates at any single temperature[1]; low temperatures with their associated high humidity favour prolongation of dormancy. If dormant seeds are placed into water immediately after abscission from the parent plant and are kept at 20°C dormancy will persist for several years. Conversely, after-ripening of dry seeds at high temperatures is accelerated compared to low temperatures[3]. Moist seeds kept at high temperatures (above 23°C) take on secondary dormancy[35] and this is accentuated under anoxia. The depth of this secondary dormancy is inversely proportional to the length of the after-ripening period and only seeds that show primary dormancy can be induced into secondary dormancy. The induction of secondary dormancy by high temperatures is qualitatively different from the state induced just by anoxia.

Light.   Wild oats show the classical reversible phytochrome response to red/far-red light at low fluence rates, and inhibitory responses to prolonged white, far-red and blue light of high fluence rates (High irradiance reaction (HIR))[11,12]. Past confusion in the literature about the effects of light on wild oat germination is undoubtedly due to considerable genetic diversity of response to both daylength and temperature during seed development and at the time of potential germination.

The effects of light during the time of potential germination can only be demonstrated when dormancy is almost lost through after-ripening, or in genotypes that have no innate dormancy provided they are pre-conditioned through the application of water stress. The ultimate effects of light are on the embryo and are particularly dependent on the water status of the seed[11]. Hulls of different colour can mediate changes in the light quality reaching the embryo (Hou,J.Q. & Simpson,G.M. unpublished data).

Photoperiodism.   There is genetic diversity within wild oats for the timing of flowering in response to photoperiod. Long days shorten the time to flowering and short days delay flowering and time to maturation thereby increasing the level of seed dormancy[32]. Thus wild oat seed transferred to different latitudes as contaminants of wheat or barley crops can show changes in the level of seed dormancy in the next generation.

Gases.   Increasing oxygen partial pressure above the normal atmospheric level increases germination of dormant seeds[33] and anoxia prolongs dormancy, or induces secondary dormancy through changes in the metabolism of the embryo. Substances that are normally respiratory inhibitors (cyanide, ethanol, nitrate ion, azide) can break dormancy by influencing the activity of cytochrome transfer of electrons to oxygen. Some organic acids can break dormancy at particular stages of after-ripening and all of the above effects involve interactions with the oxidative metabolism of the seed. An absence of carbon dioxide in the germination environment with partially-dormant seeds increases the light-promoted inhibition of germination[10] and increasing concentrations of carbon dioxide cause the light reaction to disappear. Ammonia[8] and aluminium phosphide[6] can overcome dormancy by stimulating respiratory activity. There is a great deal of evidence to indicate that loss of dormancy is associated with significant changes in respiratory activity in both embryo and endosperm tissues[34].

Growth regulators.   Many different substances with growth regulator activity have been tested for their ability to remove seed dormancy in the wild oat[9]. The most effective substance is gibberellic acid (GA3). It can prevent the induction of dormancy in the developing seed[25] and overcome primary and secondary dormancy of mature seeds[34]. Not all forms of dormancy can be overcome by GA3 and in genotypes with these forms some after-ripening is needed before the promoting effect of gibberellin is seen[37]. Several inorganic (e.g. nitrate) and organic substances (e.g. organic acids and ethanol) can promote the loss of dormancy in some, but not all, genotypes of the wild oat[34]. Gibberellin occurs naturally in the wild oat seed and plays an active role in the natural loss of dormancy via the influence of environmental factors. Natural levels of nitrate in the soil or application of fertilizer nitrate can initiate the loss of dormancy in seeds buried in the seed bank. Differences in soil texture (e.g. sandy loam or heavy clay) have less effect on altering the expression of dormancy than differences in water availability and depth of the water table[20].

Agricultural practices.   The practice of summer-fallowing can lead to the selection of wild oat populations with enhanced levels of seed dormancy[17]. Herbicides such as diallate and triallate also cause shifts in genetic constitution of wild oat populations as herbicide resistance develops[16]. Mortality of wild oat seeds is high on an undisturbed soil surface[5] but continued exposure to light can induce deep dormancy in those seeds that survive[12]. Seeds buried to considerable depth survive longer than those near the surface[19] with a probability of sustaining dormancy because of the moist environment.

Modelling Wild Oat Seed Dormancy

A useful way of taking account of the nature of dormancy, in terms of a systems description, is to look upon each structural and environmental factor that can influence dormancy as being able to potentially affect the development of the embryo[34]. Many experiments have shown that dormancy is lost in a mature autonomous seed through a series of sequential steps, driven by environmental factors, that ultimately lead to germination. By contrast, a number of the same factors are equally capable of inducing dormancy during the differentiation and maturation of the seed on the parent plant. The explanation of these opposing effects by the same factors at different times in the life of the seed seems related to the need for the different functional parts of the seed to be synchronized at each progressive stage of development.

Failure to achieve synchronization means failure to germinate but not necessarily a cessation of further development in all components of the seed structure and function. Some minimum number of components of the seed are required to be able to operate together in concert before the rapid phase of growth, called germination, can occur. Germination can be made to occur in the earliest stages of zygote growth provided the correct balance of driving factors is present. Gibberellin has a synchronizing influence on carbohydrate metabolism, and light and temperature each have synchronizing effects, both within individual seeds, and among a population of seeds. I believe that asynchronism of active physiological processes is a reasonable explanation for the failure of germination that we call 'dormancy'. I have tried to depict this with three-dimensional models[34].

I have concluded, after working for more than 33 years on problems of seed dormancy in the wild oat, that attempts to directly control seed dormancy, as a means of limiting the weed problem, are likely to be frustrated simply because of the many ways that dormancy can be expressed. No single method of interference could eliminate every expression of dormancy in such a genetically heterogenous species. Because a high proportion of the seeds shed in any one year are likely to be non-dormant or not able to retain viability for very long in the field, the proportion of seeds that express dormancy for more than three or four years is likely to be quite small. The proportion of these dormant seeds that can remain viable for longer than six or seven years is likely to be very, very, small. Thus while dormancy ensures survival for several years of a small proportion of the shed seed in one year it is clearly the total number of seeds shed that will determine the size of the soil seed bank and the potential number of emerging seedlings in any season. The work of Medd[23] suggests that reducing fecundity is the single most important step in a long-term plan for control of wild oats. As with most control measures in agronomy I would expect that integration of a number of control steps is likely to be necessary. For example avoid build-up of herbicide resistant strains, prevent the development of specific types exhibiting deep dormancy from summer-fallow practice and avoid deep ploughing that prolongs dormancy and viability. The wild oat has persisted since biblical times and flourished in this century despite the use of at least 11 quite effective herbicides. Dormancy is clearly a major factor in the long-term persistence of this very troublesome weed.

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Developments in the Control of Wild Oats: Chemical Options

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Summary

Wild oats are recognised as serious problem weeds on a global basis and currently over 10% (>$1,240 million) of the total herbicide sales are directed at wild oats and associated weeds. The majority of selective avenacides are used in cereals and the most recent introductions are clodinafop, fenoxaprop-ethyl (both with safeners) and tralkoxydim, which are all post-emergence compounds. *Avena* control is also practised in broadleaf crops with compounds like fluazifop-P-butyl, and non-selective compounds such as paraquat and glyphosate are used to control the full weed spectrum including *Avena*. Exploitation of chemical control options will, however, not be restricted to the introduction of new active ingredients, and significant progress will be made through the use of novel adjuvants and safeners.

Introduction and Scope

Wild oats have been recognised as problem weeds for centuries, but it is only in the last 40 years that the situation has become serious on a global basis and that agrochemicals have been used extensively to control them. It is impossible to give a comprehensive review of chemical control options in a paper of this length covering selective and non-selective control, and pre-emergence and post-emergence applications. Therefore, this paper will mention all areas, but will focus primarily on cereal-selective *Avena* control, particularly for *Avena fatua*, *Avena sterilis* and *Avena ludoviciana*.

Evolution of Chemical Control of *Avena* Species

The first chemicals used commercially for wild oat control were non-selective grass killers such as TCA and propham which were applied at rates ranging from 2 to 8kg/ha. These compounds, however, were damaging to cereals, and it was not until the late 1950's that the earliest selective compounds — barban and diallate — were introduced. Over the past thirty years, research has concentrated on discovering and developing further selective wild oat killers to add to the farmers' armoury. The successful introduction of compounds like flamprop-methyl, fenoxaprop-ethyl, imazamethabenz-methyl and most recently tralkoxydim, has allowed chemical application rates for wild oat control to be reduced to the 60 to 600g/ha range, and further research will probably permit even lower levels of application in the future.

Chemical Usage for the Control of Wild Oats

The global herbicide market in 1991 was estimated to be $11,905 million[2], but it is extremely difficult to quantify the herbicide inputs used for controlling wild oats. We estimate that in cereals, sales of products for the control of wild oats plus other weeds amount to $740 million per annum (see Figure 1), and is dominated by post-emergence treatments. In addition, substantial markets exist for wild oat control in other sectors, for selective (e.g. fluazifop-P-butyl) and non-selective (eg. glyphosate, paraquat) uses, and we estimate these to be in excess of $500 million each year. Thus, on a global basis, over *10%* of all herbicide inputs are aimed at controlling wild oats.

Total Market Value = $2,300 million
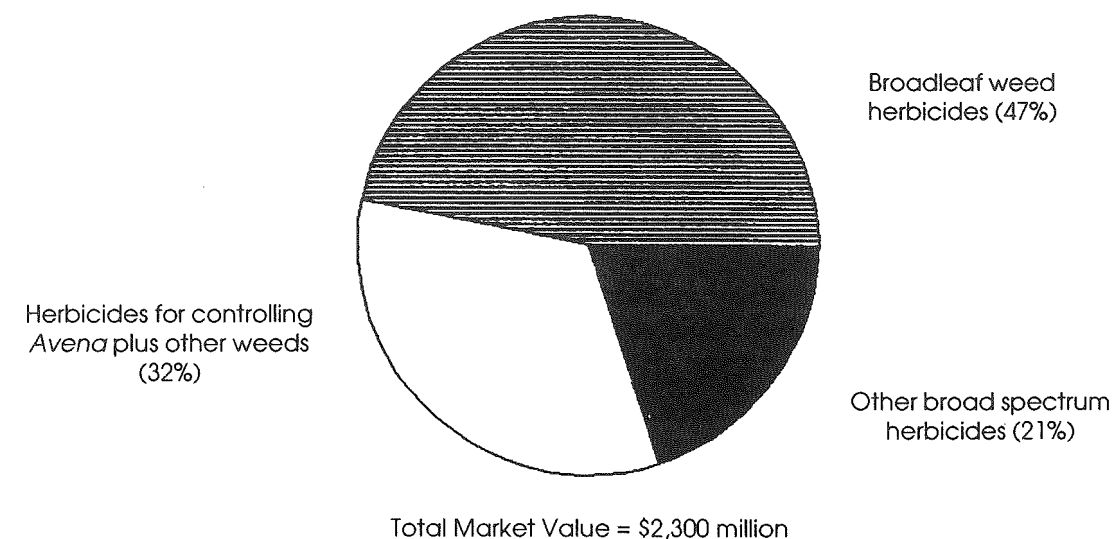
Figure 1. Global Cereal Herbicide Market Divided on an End Use Basis (adapted from ref 2)

Pre-Emergence Cereal Selective Avenacides

The major pre-emergence avenacides are diallate, triallate and trifluralin. All of these compounds were introduced in the 1960's and relatively high rates are required for wild oat control (typically 1–4 kg a.i./ha) compared to their more recent post-emergence counterparts. Other compounds such as chlorotoluron and isoproturon control a wide range of weed species, including *Avena*.

Trifluralin was introduced by Eli Lilly & Co in 1960 under the code name Treflan® and has found widespread use in a range of cereal and broadleaf crops. The compound belongs to the dinitroaniline class of chemistry and is particularly active on both warm climate and temperate grass weeds, together with a variety of broadleaved weeds at rates of 0.5–1.0 kg a.i./ha.

The thiocarbamate herbicides, diallate and triallate, were introduced by Monsanto in 1960/61 as Avadex® and Avadex BW®, respectively. Like trifluralin, both are volatile and require incorporation into the soil where they are particularly active against wild oats at rates of 1.5–2.0 kg a.i./ha.

Thus, the pre-emergence market for controlling *Avena* species in cereals is dominated by "old" compounds, and little has changed in recent years regarding new product introductions or significant changes in market share.

Post-Emergence Cereal Selective Avenacides

The post-emergence market for controlling *Avena* species has, in contrast, changed markedly in recent years, and as Hutson and Roberts[5] have reviewed the area of post-emergence wild oat herbicides up until 1987, this paper will only focus on those herbicides which have either appeared since that time or have taken significant market share in the intervening period. Table 1 lists the major cereal selective graminicides, both recent and established, together with their use rates and spectrum (see Figure 2 for selected structures).

Table 1. Major Cereal Selective Graminicides

Trade Name	Common Name	Company	Chemical Class [1]	Spectrum (Genus)	Rate (g./ha)
Puma® Cheetah®	Fenoxapropethyl	Hoechst	AOP	Avena Alopecurus Setaria Phalaris	60-180
Hoegrass® Hoelon®	Diclofop-methyl	Hoechst	AOP	Avena Lolium Setaria	700 - 1000
Topik®	Clodinafop (proposed)	Ciba-Geigy	AOP	Avena Alopecurus Setaria Phalaris	40 - 80
Grasp® Achieve®	Tralkoxydim	ICI	CD	Avena Alopecurus Setaria Lolium Phalaris	150-350
Assert® Dagger®	Imazametha-benzmethyl	Cyanamid	IMD	Avena Alopecurus Broadleaf weeds	400-800
Avenge®	Difenzoquat metisulfate	Shell	DPP	Avena	500-1400
Mataven® Commando®	Flamprop-methyl	Shell	BA	Avena	400-600

[1]
AOP	:	Aryloxyphenoxy-propanoate	CD :	Cyclohexanedione
IMD	:	Imidazolinone	PYR :	Diphenylpyrazolium
BA	:	Benzolyl-alaninate		

Details of two types of cereal selective herbicides — ACC'ase inhibitors and ALS inhibitors — are presented.

Figure 2. Structures of some Key Cereal Selective Graminicides —
(1) Fenoxaprop-ethyl, (2) Fenchlorazole (Safener),
(3) Tralkoxydim, (4) Difenzoquat

ACC'ase Inhibitors

Diclofop-methyl (Hoegrass®) was the first example of a new class of herbicides which specifically controlled grass weeds. This compound, introduced by Hoechst in 1975, was subsequently found to inhibit the enzyme acetyl co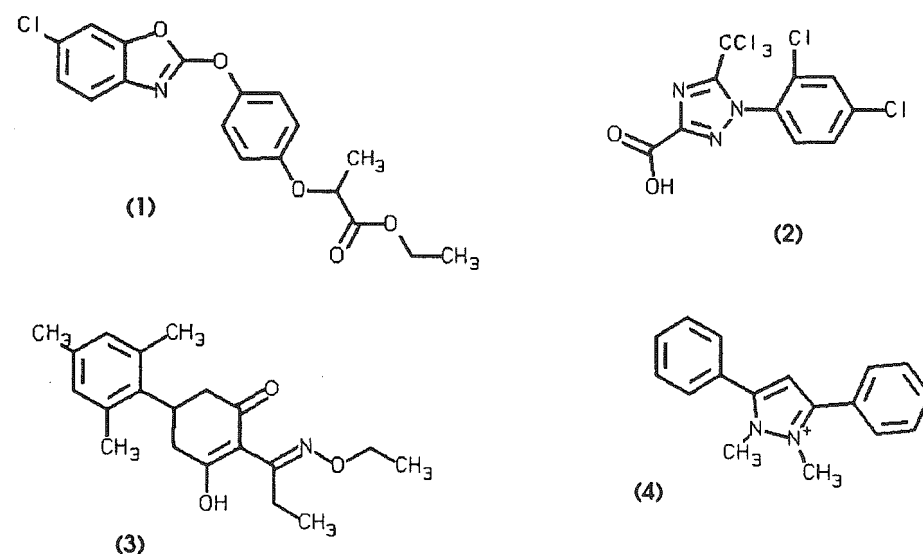enzyme A carboxylase (ACC'ase) which is involved in regulating fatty acid biosynthesis[9]. A new generation of broadleaf crop selective graminicides followed this discovery with the introduction of fluazifop-butyl by ICI; this area will be discussed further later in this review. Cereal selectivity amongst ACC'ase inhibitors has proved to be difficult to achieve, but three recently introduced products are used for Avena control in cereals — two aryloxyphenoxy-propanoates used in combination with safeners, and one cyclohexanedione with inherent selectivity.

Fenoxaprop-ethyl (Whip®) from Hoechst is an example of a broadleaf selective aryloxyphenoxy-propanoate which is active on a wide range of warm climate and temperate grass weeds and is sold into a variety of broadleaf crops such as soya, cotton and canola. The herbicide can also be used in graminaceous crops such as rice and wheat, but selectivity can be marginal. The development of the safener fenchlorazole-ethyl greatly increased the level of tolerance in wheat to fenoxaprop-ethyl, without reducing weed control, and this mixture (4:1 ratio of herbicide to safener) has been used extensively since its introduction as Puma® in 1989[4]. Fenoxaprop-ethyl is registered for use in winter and spring wheat, durum wheat, triticale and rye — but not barley, where the crop phytotoxicity is too high. Fenchlorazole-ethyl is believed to improve selectivity in wheat by increasing the rate of metabolism of the herbicide to inactive degradation products[11], whereas metabolism does not appear to be as extensive in grass weeds[13].

Clodinafop from Ciba-Geigy is another aryloxyphenoxy-propanoate graminicide which is mixed with a safener (cloquintocet) to confer tolerance in wheat, since the graminicide itself is too damaging to the crop. Reference to Table 1 shows that the spectrum of Topik® (safened clodinafop) is similar to that of fenoxaprop-ethyl, giving excellent control of a number of important grass weeds such as Avena fatua, Alopecurus myosuroides and Setaria viridis. As with fenoxaprop-ethyl, the safener stimulates the rate of metabolism of the graminicide[6] in wheat, and to a lesser extent in barley, which explains why barley tolerance is marginal.

Tralkoxydim from ICI is the only cyclohexanedione which is selective in small grain cereals. Discovered by ICI Australia and introduced in 1987 as Grasp®[12] it is unique in that it is the only ACC'ase graminicide which is safe for use on both wheat and barley, including winter and spring varieties, durum and hard red wheat spring varieties, triticale and rye. The excellent cereal selectivity is believed to be due to the rapid and extensive metabolism of the herbicide in the crop (Hadfield, ICI unpublished data), although uptake and translocation may also play a role since both are significantly greater in wild oats than in wheat (Bartlett, ICI unpublished data).

Like fenoxaprop-ethyl and clodinafop, tralkoxydim has a wide growth stage window of application with respect to the crop and grass weeds, and is particularly effective against wild oats, rye grass and foxtails. The cyclohexanedione chemistry is highly responsive to both formulation and adjuvant technology and significant advances have already been made in both areas, for example with the introduction of a "water dispersible granule" (WG) formulation in Canada as Achieve®. Such advances offer many advantages in terms of operator safety, ease of use and more readily disposable packaging, together with reduced application rates and consistency of weed control under a range of environmental conditions.

ALS Inhibitors

Acetolactate synthase is the target site of the sulfonyl ureas, imidazolinones and the triazolo-pyrimidine group of herbicides. To date there is only one example of a wild oat graminicide which is cereal selective from within these chemical groups and that is imazamethabenz-methyl or Assert® from Cyanamid. This compound was launched in 1982[10] and since that time has taken a significant market share from the older, and more established wild oat herbicides. Imazamethabenz-methyl is selective in both wheat and barley and is particularly effective against wild oats and blackgrass. However, unlike the ACC'ase graminicides, it is also active on a number of broad leaved weeds such as Brassi

cacae species. Selectivity is thought to be due to oxidation and subsequent glucose conjugation in wheat, whereas the toxic free acids are formed in wild oat[1].

Other Situations Requiring *Avena* Control

Control of *Avena* spp. is important in two other situations. The first of these is for selective control in crops other than cereals and the second is for non-selective uses. The former sector includes compounds like fluazifop-P-butyl, quizalofop-P-ethyl and haloxyfop, while the latter encompasses paraquat dichloride, glufosinate ammonium, glyphosate-isopropylammonium and glyphosate-trimesium (Table 2 and Figure 3).

Table 2. Examples of non-cereal selective Herbicides used for controlling *Avena* spp

Trade Name	Common Name	Company	Chemical Class [1]	Spectrum (Genus)	Rate (g./ha)
Fusilade®	Fluazifop-P-butyl	ICI	AOP	*Avena* *Alopecurus* Other grasses	188-375
Gallant® Verdict®	Haloxyfop-etotyl Haloxyfop-methyl	DowElanco	AOP	*Avena* Other grasses	100-300
Targa® Assure®	Quizalofop-P-ethyl	Nissan Du Pont	AOP	*Avena* Other grasses	25-250
Gramoxone®	Paraquat dichloride	ICI	B	All plant species	140-2210
Basta®	Glufosinate-ammonium	Hoechst	PI	All plant species	1000-2000
Roundup®	Glyphosate-isopropyl-ammonium	Monsanto	PA	All plant species	340-2240
Touchdown®	Glyphosate-trimesium	ICI	PA	All plant species	340-2240

[1]
AOP	:	Aryloxyphenoxy-propanoate	B	:	Bipyridyl
PI	:	Phosphinic acid	PA	:	Phosphonic acid

The majority of these compounds are well established, particularly Fusilade® which was introduced in 1981 for the control of *Avena* and other grass weeds in broadleaf crops, such as sugar beet and potatoes. The product acts as an ACC'ase inhibitor and is quickly absorbed through the leaf surface, hydrolysed to the active acid and translocated through the phloem and xylem. In the case of *Avena* species, it accumulates in the meristems. Three of the non-selective compounds are also widely used — paraquat was introduced by ICI in 1959 and destroys green plant tissue mainly by contact action and some translocation, and is rapidly inactivated on contact with soil; glufosinate is a contact compound launched by Hoechst in 1984, which is translocated within the leaves; and glyphosate, which was introduced in 1971 by Monsanto, is absorbed by the foliage and stems and translocated throughout the plant.

The most recent introduction to this market is glyphosate-trimesium, a systemic, non-selective herbicide which is fairly fast-acting and has good rainfastness properties.
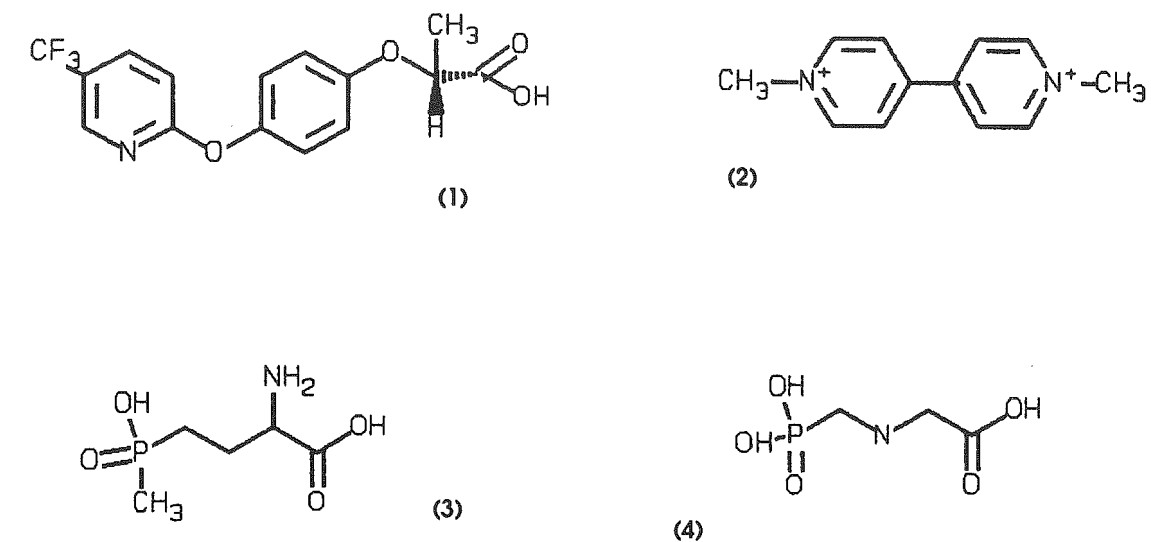
Figure 3. Structures of some Key Non-cereal Selective Herbicides used for controlling *Avena* — (1) Fluazifop-P, (2) Paraquat, (3) Glufosinate-ammonium, (4) Glyphosate

Future Chemical Options for Wild Oat Control

Since the Hutson and Roberts review[5] of wild oat herbicides in 1987, several new and exciting compounds have been developed and commercialised to control this weed, both selectively and in non-crop situations. Some of these herbicides, like tralkoxydim, safened clodinafop and glyphosate-trimesium, are still in a relatively early stage of sales and development and are only now just entering major markets where they will offer significant advantages for growers over the established products.

Advances in adjuvant and formulation technology will continue to enhance the activity of existing herbicides in controlling wild oats by reducing the rates of active ingredients required for activity and/or providing more robust treatments to cope with environmental stresses such as drought. Examples of success include BASF's adjuvant Merge®, and ICI's new adjuvant "TF8035" for tralkoxydim. "TF8035" can, in fact, be used at lower rates than the presently recommended crop oil concentrate (COC), and could facilitate the effective use of less tralkoxydim than previously recommended[7].

New safeners, such as cloquintocet and fenchlorazole-ethyl, continue to be developed to improve crop tolerance to graminicides, and it is likely that more will follow. Herbicide mixtures for wild oat control are also likely to increase in the future. This has certainly been the case in the broadleaf weed sector where growers have adopted a "mix-and-match" approach to weed control, but this has not been seen to any great extent in the graminicide sector. However, as more avenacides become available, mixing may become more attractive to growers, not only to complement their grass weed spectrum but also as a means to manage herbicide resistance.

Apart from the new herbicides discussed above there are relatively few novel compounds in development targeted for wild oat control. The Brighton Conference in 1991 saw the introduction of only one new avenacide, NC-330 from Nissan — a sulfonyl urea for blackgrass and wild oat control in small grain cereals[8]. However, more recent information suggests that Nissan are unlikely to develop this compound further[3]. Overall, though, new products for *Avena* control will be required in the future, alongside improved use strategies if we are to guarantee good levels of control, particularly if resistance to graminicides occurs.

In recent years considerable progress has been made in the area of herbicide resistant crops. Companies like ICI Seeds have now commercialised herbicide tolerant crops, such as IT Corn® which is tolerant to high rates of the imidazolinone herbicide imazamethapyr, while others are developing crops which are resistant to a range of herbicides, such as glufosinate, glyphosate and sulfonyl ureas. In small grain cereals one US group has already had success with isolating mutants of wheat and barley which are resistant to the ACC'ase graminicides, while others (at the University of Florida working with Monsanto) have succeeded in introducing glufosinate resistance into wheat[11]. This subject will be covered in detail later in this meeting, but the advent of such varieties will obviously increase the number and range of chemical options open to growers in controlling wild oats in the future.

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New Developments in the Control of Wild Oats: Australian Advances

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Summary

The impact of selective wild oat herbicides in Australia is reviewed in terms of their ability to reduce weed density, increase crop yield, their impact on wild oat seed production and their ability to contain wild oat populations. Directions for technological and economic advances which will improve the management of wild oats in Australia are suggested and discussed briefly.

Introduction

Few weeds of winter cropping systems in Australia are more important economically than wild oats (*Avena* spp.) They occur throughout the Australian winter cereal belt, causing losses conservatively estimated at $42 million in wheat during 1987[14] due to competition reducing crop yields and to the cost of herbicides applied to control them. A recent survey of farmers interviewed by Hoechst Australia Ltd. found that wild oats occur on two out of three farms throughout winter cereal growing areas, are increasing on over 40% of the infested farms and the managers of more than one third of these farms find it difficult to control wild oats (P. Howat, pers. comm. 1990). The difficulty in controlling wild oats in wheat had likewise been shown in previous farmer surveys reported by Martin and McMillan[6] and Martin *et al.* [7].

This paper reviews the efficacy of wild oat herbicides, puts some current advances into context and examines opportunities for improving the management of wild oats in Australia.

Control of Wild Oats in Australia

Selective wild oat herbicides first appeared in Australia in the late 1950's. This section reviews the impact of these herbicides in terms of their ability to reduce wild oat plant density in the crop, increase crop yield, their impact on wild oat seed production and their ability to contain wild oat populations. The review collectively examines data published by Martin *et al.* [9], Reeves *et al.* [26], Wilson[31,32], Wilson *et al.* [34] and unpublished data provided by Martin and Felton (pers. comm. 1992) and Wilson (pers. comm. 1992). Some above and some sub-label dose rate results have been included in the data base and it should be emphasised that interpretations drawn from these retrospective data do not necessarily reflect the performance of recently registered herbicides.

Plant control

Since competition is a function of plant density, a primary objective in weed control is to kill plants. Irrespective of the type of herbicide used, there is wide variation in the level of control achieved for any given density (Figure 1). Wild oats increased in density in some treatments (i.e. negative plant control, where points lie above the line of unit slope), indicative that seedlings must have emerged after treatment. In most instances, however, there were fewer plants after treatment. Staggered seedling recruitment is a feature of wild oats[24,29], which undoubtedly accounts for part of the variation in plant control (and yield response, see below). The variability in plant control is further illustrated in Figure 2 but, more importantly, the graph demonstrates that a high level of control (say 80%) can be achieved under some conditions by applying herbicides anytime from pre-emergence through to late post emergence, regardless of plant density. The converse result of poor control is also independent of plant density.
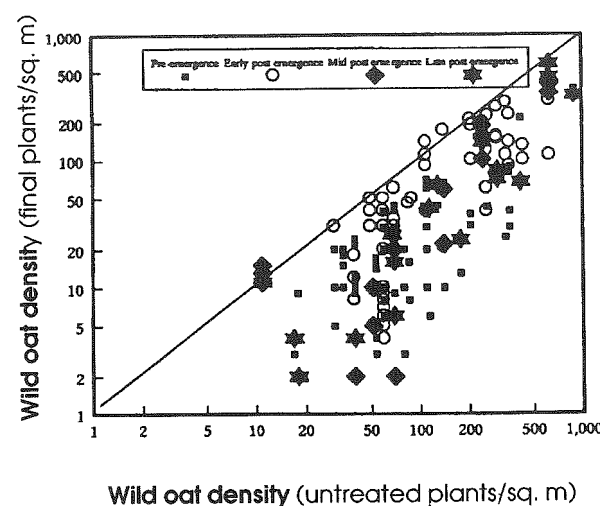
Figure 1. Relationship between untreated and final wild oat plant density in crops treated with pre- or post emergence herbicides (log scale).
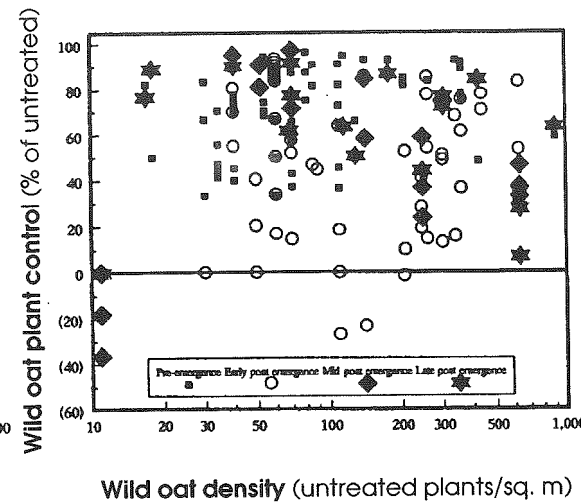
Figure 2. Relationship between untreated wild oat plant density and plant control (% of untreated) in crops treated with pre- or post emergence herbicides (semi log scale).

Yield response

There is little doubt in Australia that wild oats compete strongly with crops, as illustrated by Martin *et al.* [5] and Poole and Gill [20]. Consequently, the compelling reason for using herbicides is to protect crops from the competitive impact of weeds. The scatter diagram of the proportional increase in yield against the proportional reduction in plant density provides one measure of herbicide efficacy (Figure 3). Under some circumstances crops failed to give a positive yield response after treatment with wild oat herbicides, despite providing high levels of control. Many other treatments, regardless of the time of application or level of control achieved provided 50% increase in yield, whereas the highest yield responses were only realised above 50% plant control. Clearly, yield response is poorly related to herbicide efficacy.

Yield increases varied enormously from negative responses, regardless of wild oat density, up to a 2.4 t ha^{-1} increase when wild oats were reduced to low densities (Figure 4). Assuming a net price of $140 per tonne for wheat, an additional ⅓ tonne of grain is required to recoup the cost of most wild oat herbicides (i.e. $40 for herbicide and $5 ha^{-1} application cost). Based on these data there is a high risk of failing to achieve this goal, irrespective of wild oats density (Figure 4).
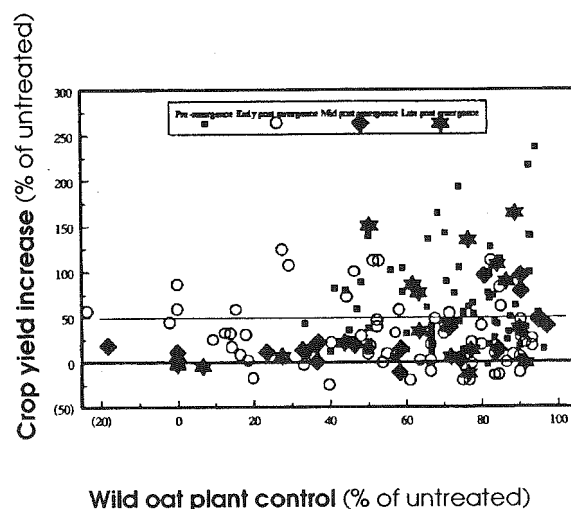
Figure 3. Relationship between wild oat plant control (% of untreated density) and crop yield increase (% of untreated control) in crops treated with pre- or post emergence herbicides.

Figure 4. Relationship between final wild oat plant density and actual crop yield increase (kg/ha) in crops treated with pre- or post emergence herbicides (semi log scale).

Of all the groups, pre-emergence herbicides tended to be slightly more consistent in providing yield increases above this cost threshold. This is consistent with the knowledge that wild oats which emerge with the crop tend to be the most competitive, particularly at high densities, thereby favouring early removal [4].

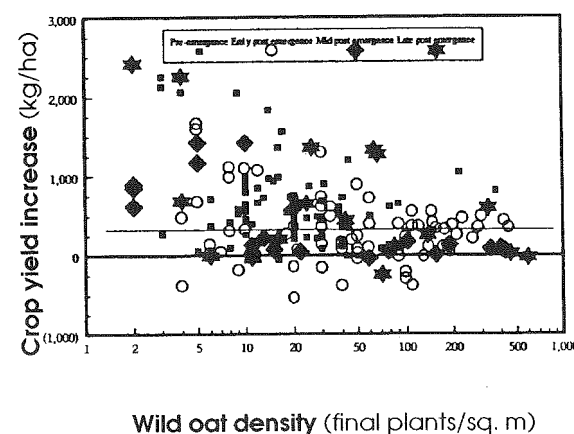Unquestionably, the reliability of wild oat herbicides needs to be improved, especially for low weed densities. I contend that far too much emphasis has been placed on forecasting yield loss to decide whether or not to spray, at the expense of determining better decision rules based on spray responses. Dose response curves detailing the response of herbicides under a range of environmental conditions would seem to be of greater use for decision making than simple thresholds [16] and having this knowledge would remove some of the risk in spray decisions.

Figure 4 further shows that there is a greater chance of achieving highest yield increases by reducing wild oats to low densities, as would be expected. Moreover, the possibility of recovering the cost of herbicides appears to become more risky when higher densities of plants remain following treatment. This, together with the expectation that wild oats cause greatest absolute loss at high densities, indicates that treatment becomes increasingly futile as the density of infestation increases, unless extremely high (say 95%) plant control is achieved. As a guide, therefore, my suggestion is that profitable management of wild oats becomes riskier when expected final wild oat densities exceed crop density, and where this occurs such fields should perhaps be rotated to other pursuits.

As an aside to the thrust of this paper, it cannot be emphasised too strongly that agronomic aspects such as choice and density of crop and crop cultivar, time of planting, relative time of crop and wild oat emergence, fertiliser requirements and placement all impact substantially on competition and yield loss from wild oats, as discussed by Barton *et al.* [1] and Medd [10]. My feeling is, however, that we can make substantial gains in these regards and more emphasis should be placed on **getting the agronomy right** to optimise the management of wild oats in Australia. Optimising crop density should be given highest priority.

Seed production

Being annuals, wild oats depend on their seeds for survival, multiplication and invasion, but the processes regulating seed production have been largely ignored in the management of wild oats. The number of seeds produced per plant (fecundity) is clearly one of the most plastic plant parameters, as shown in Figure 5. Maximum fecundity was around 230 seeds plant^{-1}, with output falling sharply to mostly below 50 seeds plant^{-1} for densities above 50 plants m^{-2} (Figure 5). Seed production by untreated plants ranged from 1,000 to a peak of around 10,000 seeds m^{-2} (Figure 6). In herbicide treated crops, seed production was reduced with a ceiling of around 5,000 and minimum of 300 seeds m^{-2} for densities above 50 plants m^{-2}.

It is widely held that one of the main failings of wild oat herbicides is their inability to control seed production [6,18,31]. These data mostly support this generalisation, but it is surprising to find that herbicides mostly suppressed seed production moreso than plant density (Figure 8). Furthermore, many pre-emergence treatments and some others (those lying below the line of unit slope) illustrate that some plants, presumably those which survive being treated, or which are recruited after treatment, respond by becoming more fecund (Figures 7 and 8); possibly because they face less competition. Nevertheless, plant density is rarely brought below 5 plants m^{-2} from which 100 seeds m^{-2} are mostly produced (Figure 6). There are indications, however, that better control of seed production is attainable. Up to almost 100% control of fecundity has sometimes been achieved, regardless of density, with late post emergence herbicides (Figure 7). This tactic has recently been corroborated by Medd *et al.* [12] and is currently being further tested in northern NSW. The concept of controlling seed production generally deserves greater attention, as amplified below under demographic strategies.

Fecundity of wild oats is clearly density dependent. Not only does seed output decline exponentially as density of the weed increases (Figure 5), but it also responds to crop density. Radford *et al.* [25] showed that whilst seed production was maximal for all weed densities at low crop densities, it declined as crop density increased, especially at low weed densities (see [10] for response surface of these data).
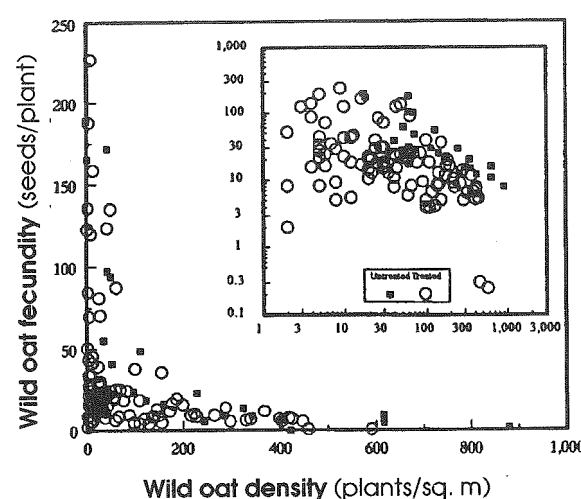
Figure 5. Seed production per plant (fecundity) for wild oats which have matured in either herbicide treated or untreated crops, plotted as a function of plant density. Inset depicts data using log scale.
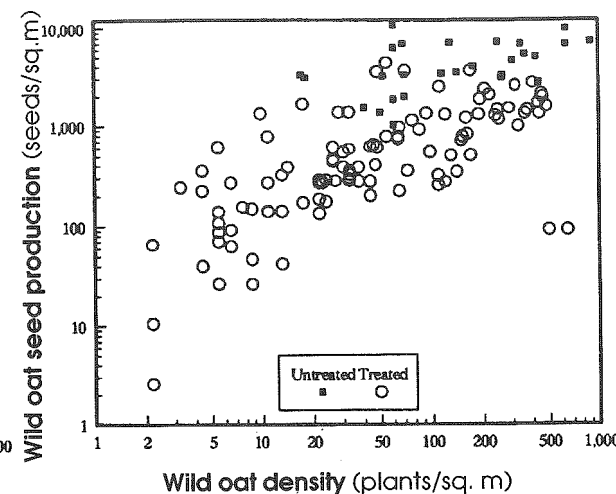
Figure 6. Seed production per unit area for wild oats which have matured in either herbicide treated or untreated crops, plotted as a function of plant density (log scale).
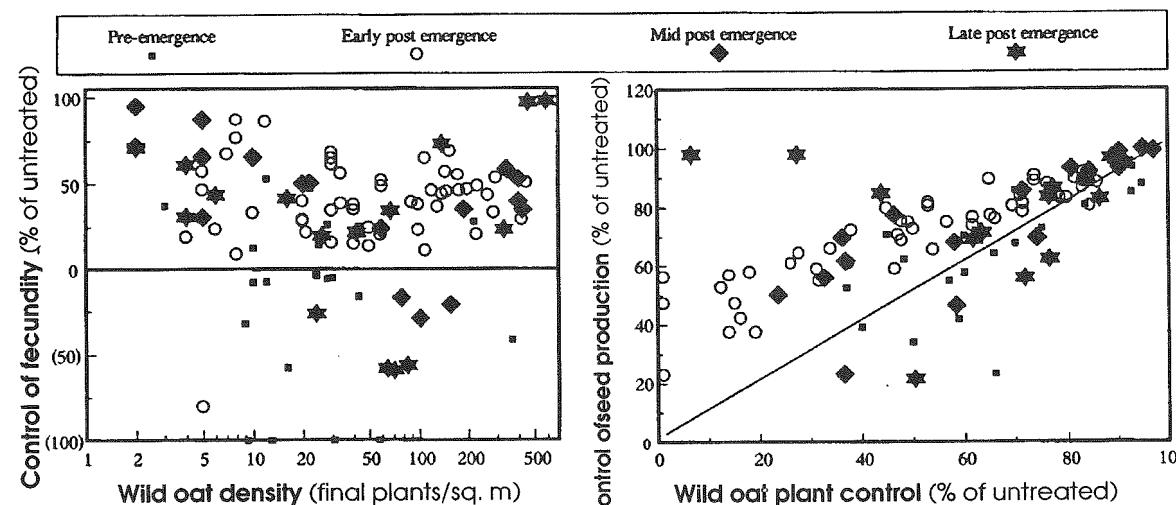
Figure 7. Relationship between the control of seed production per plant (% of untreated plants) and final plant density for crops treated with pre- or post emergence herbicides (semi log scale).
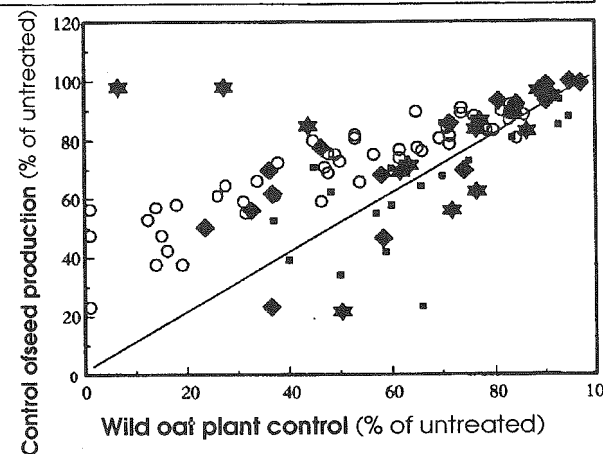
Figure 8. Relationship between the control of seed production per unit area (% of untreated plants) and plant control (% of untreated plants) for crops treated with pre- or post emergence herbicides (semi log scale).

Population containment

Modern farmers have been preoccupied with treating weed infestations to maximise profits in the current year. Few farmers consider the consequences such tactics have on populations in subsequent years. Since herbicide use decisions made in the current year can have impacts in the future (e.g. herbicide resistance), a change in attitude is needed so that weed populations are managed in a long term framework.

Continuous cropping. Although partially effective in reducing competition, both research and grower evidence has highlighted the ineffectiveness of herbicides in preventing the build-up of wild oat populations. Seed banks of wild oats increased four to six-fold over a three year continuous wheat cropping cycle using diclofop-methyl[11] and up to twelve-fold using flamprop-methyl or tri-allate[8]. The population increases in both studies occurred irrespective of whether conventional or conservation tillage techniques were used to prepare seedbeds and typify the failure of management systems to contain wild oats in the continuous cropping systems practiced throughout much of the winter cropping areas.

One explanation why wild oats thrive in continuous wheat systems is that they are seemingly selectively stimulated by decomposing wheat residues[22]. Growth and seed production of

wild oats grown in monoculture under field conditions were increased 10 and 42 fold respectively in the presence of wheat crop residues[23].

Rotational cropping. A highly successful strategy to control wild oat populations is to prevent seed production by clean winter fallowing. Philpots[19] found wild oat infestations were reduced from 93.3 plants m^{-2} to 3.2 and 0.2 plants m^{-2} after one or two consecutive years of winter fallow. Fallowing for a third year gave no additional decrease in wild oat density. Wilson et al.[34] obtained similar results and Martin and Felton[8] demonstrated that no tillage winter fallowing techniques were just as effective as conventional tillage practices. Wilson[32] further demonstrated that where summer crops could be grown, control of wild oats by winter fallowing could be achieved without cost, negating substantial financial losses likely in continuous wheat. Sorghum (*Sorghum bicolor* (L.) Moench) is not only the summer crop option widely preferred but it would appear to facilitate control of wild oats by delaying and contracting its emergence pattern[3,21].

Although there are no data specific to Australian conditions, Thurston[6] and Wilson and Phipps[33] found in England that in pasture rotations, seed banks decline rapidly in the first year and slowly thereafter. Although the small numbers of seeds which persist for several years are sufficient to re-establish infestations in ensuing crops, every effort should be made to minimise seed banks by ensuring that seed production does not occur in such rotations.

Strategies for Improving Control

Clearly from the above overview, weaknesses in existing control tactics have to be overcome if more efficient control strategies are to be developed. The overwhelming evidence indicates that wild oat persistence within crops is due more to the input of new seed, rather than to seed longevity in the soil (as is widely believed). Seed dormancy can allow seeds to accumulate in the soil[2,27]; however, proliferation of wild oat populations through long term seed bank accumulation seems highly unlikely given that its seeds are generally short-lived[24,29] and seed banks decline annually at a rate of 70% or greater[11]. The work with summer crop rotations[e.g.8,19,22,32], weighs strongly against the dormancy/longevity/carry-over mechanism, given that seed banks are rapidly purged.

Demographic strategies

One way to examine the mechanisms underlying the persistence of wild oats is to simulate their demographic behaviour. The population size of wild oats is the product of the probability of seedling recruitment (germination and emergence = g), the probability of seedling survival to adulthood (= s), the number of seeds produced per adult (fecundity = F), the probability of produced seeds entering the seed bank (seed rain = R) and the probability of seeds surviving in the soil (= d) (Figure 9). Any one of these transitional probability parameters may be regulated but, since the advent of herbicides, weed control has concentrated on reducing s by maximising seedling mortality.

Using the generalised simulation model described by Medd and Ridings[13] it is possible to compare the relative impact of independently regulating the demographic parameters. In a hypothetical "base" population the parameters are set to mean values observed in field experiments conducted by the author at Orange, NSW with a notional starting seed bank of 1,000 seeds m^{-2} (Figure 9). In the model, recruitment occurs in three cohorts (only two active in this simplified case) with g = 0.45 and 0.05 for cohorts two and three respectively, and s = 0.25 (i.e. 75% plant mortality) for both cohorts. Fecundity relationships are given in the figure and all seed produced enters the seed bank (R = 1.0). Seeds which remain in the seed bank after recruitment has ceased have a probability of d = 0.35 of persisting in the soil (i.e. 65% seed decline). After one year under this "base" scenario the population rose to 1,552 seeds m^{-2}, and after five years to 5,749 seeds m^{-2} (Figure 9). Such an increase realistically reflects the population increases recorded in continuously cropped fields and experiments[6,8,11].

To contain the population to 1,000 seeds m^{-2} or below, plant mortality of cohort 2 would need to rise from 75 to > 87% (i.e. $s \leq 0.13$), seedling recruitment of cohort 2 reduced from 45 to 21% (i.e. $g \leq 0.21$) or alternatively seed rain would need to be reduced to $R \leq 0.59$. The population could not be contained by totally stopping seed carry-over alone (i.e. d = 0).
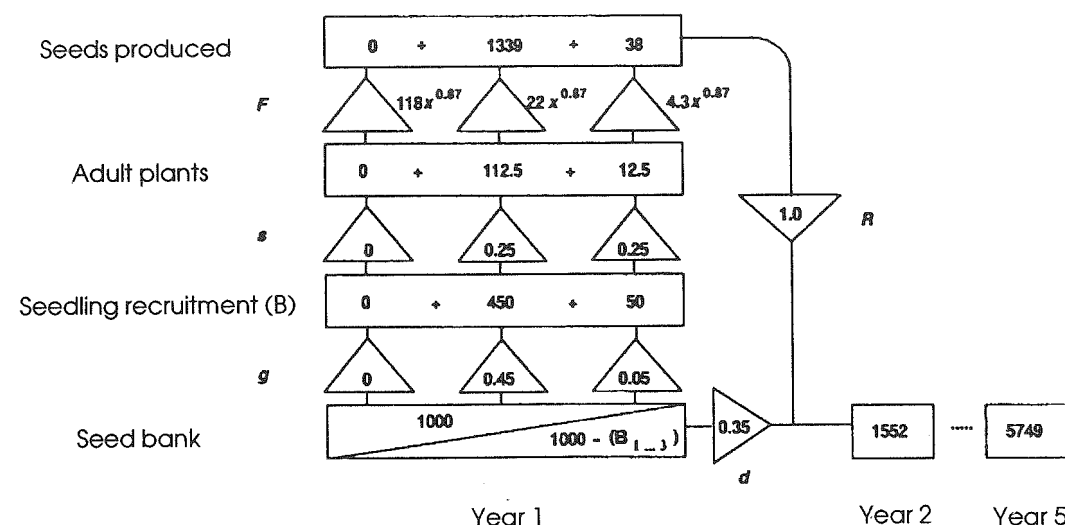
Figure 9. "Base" population dynamics model of wild oats showing three cohort streams (two active) and transitional probabilities based on field studies conducted at Orange (see [13] for details).

Population size after five years for scenarios of 50, 75 or 87.5% independent changes in *g*, *s*, *R* and *d* are given in Table 1. Increasing seedling recruitment produced by far the worst result with populations increasing exponentially by between 16 and 30 fold. Seedling survivorship of $s \leq 0.1$ or seed rain $R \leq 0.46$ would be required to contain such populations to 1,000 seeds m^{-2} or below. Decreasing seedling recruitment gave a dramatic reversal in population growth, as did reducing plant survivorship and reducing seed rain. Reducing the carry-over of seeds in the soil only marginally reduced the population compared to the base demographic behaviour.

Table 1. Population size (seeds m^{-2} in the seed bank) after five years, following separate and independent changes of 50, 75 and 87.5% in seedling recruitment (cohort 2), seedling survivorship (cohort 2), seed rain and the loss of seeds in the soil.

		Percent change in parameter		
	0	50.0	75.0	87.5
Increased seedling recruitment	5749	16230	24326	29202
Decreased seedling recruitment	5749	1105	265	84
Decreased seedling survivorship	5749	798	133	29
Decreased seed rain	5749	551	65	10
Increased seed bank loss	5749	4463	3919	3669

Technological advances

Wild oat populations, as distinct from infestations, could be better managed by developing new technology that supplements existing practices. Based on the above predictions, innovations could aim to reduce the overall pool of seed available for recruitment as an alternative to increasing seedling mortality. This could be achieved either by preventing seedling recruitment, or by reducing seed production or seed rain.

In evaluating which lines of research into wild oat control would return the greatest benefits, Pandey *et al.* [17] similarly concluded that research into decreasing seedling survival and research into reducing seed production (which effectively is equivalent to reducing seed rain) were the most profitable. Reducing seedling survival might be achieved, for instance,

through an improvement in herbicide efficacy. However, the cost of achieving such an improvement may be very high given the variability which is evident in herbicide efficacy data (see above).

Furthermore, although I have suggested above that more research into improving the reliability of herbicides is desirable, the likelihood of success in improving herbicide efficiency is questionable, considering the substantial knowledge from research already undertaken by private companies. There is also some indication that very refined application and management skills would be needed to achieve high efficacy, which could discourage adoption in low input broadacre cropping systems.

On the other hand, ways of reducing seed production are relatively unexplored. Studies might, for example, investigate ways of interrupting floral development or of preventing pollination or using selective microorganisms to predate seeds [13]. One promising method is the late application of selective post emergence herbicides, which in preliminary tests reduced wild oat seed production by up to 96% [12]. The prevention of shattering could also be explored as a means to stop seed rain. Although harvested grain would need to be cleaned, this cost would be more than offset by savings on control in future years.

Whilst it is known that deep burial of seeds is a useful way of preventing seedling recruitment [10], the prospects for innovations in this direction are not enticing. It is conceivable that chemicals which promote dormancy could be used to negatively regulate germination, but as yet none with a capacity to induce long term dormancy has been discovered [28]. Moreover, such an approach would ultimately require advances in technology which destroy seed viability so as to ensure that seeds never become available for recruitment.

Economic advances

The development of innovative technology which could be integrated with existing weed control practices would not only lead to more efficient management of wild oats, but it has been predicted as a way of reducing the overall input of herbicides, and thus lower the cost of production [15].

To fully realise benefits from such technological advances, farmers must also adopt innovative economic practices which evaluate weed control over long term horizons, as opposed to the existing approach of expecting to recover the full cost of weed control in the year of treatment (single period or current year). Using the long term investment approach, Pandey and Medd [16] estimated that Australian farmers would not only gain better control of wild oat populations, but would generate $15 ha^{-1} additional profit over five years of continuous cropping or $55 ha^{-1} over 10 years through savings on herbicide inputs.

Conclusion

In order to make advances in the management of wild oats the emphasis should be shifted from describing the yield loss impact of the weed to that of maximising yield increase from management. The concept of treating infestations should be discarded and replaced with that of managing populations in a long term framework. To achieve better control, the efficacy and reliability of herbicides needs to be improved and more attention must be given to reducing the pool of seed available for recruitment, particularly by preventing seed production or seed rain.

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The Use of Molecular Genetics in the Quest for Wild Oat Control

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Abstract

Wild oat control in cereals is possible today through the application both pre-emergent and post emergent of a range of selective herbicides. Despite the excellent control afforded by these compounds there is an increasing incidence of resistance occurring particularly to the group of products known as selective post emergent grass herbicides.

Roundup® herbicide (glyphosphate), a non selective product, readily controls wild oats at relatively low rates and, because of its unique mode of action, there is no known resistance. Monsanto scientists have introduced glyphosphate tolerance into a number of leading dicot crops using bacterium vectors. More recently this work has expanded to transformation of wheat using particle gun technology.

Aspects of the introduction of glyphosphate tolerance genes into wheat are discussed and the potential for commercialisation of this technology is evaluated.

Herbicide Resistance in Wild Oat
— The Canadian Experience

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Historical Overview

In addressing the topic of herbicide resistance in wild oat (*Avena fatua* L.), it is important at the outset to review the history of wild oat herbicide usage on the Canadian Prairies and to underscore the obvious advantages that selective wild oat herbicides have provided to Prairie producers.

Although the soil-applied wild oat herbicide, triallate, and the post-emergence herbicide, barban, were commercially available since the early 1960's, the total area treated with these products in 1972 was under 1 million hectares, or approximately 2.5% of the total area seeded to grains and oilseeds. This low use figure belied the seriousness of the wild oat problem which according to estimates at the time was costing producers more than $120 million annually[6], making it the most economically important weed on the Prairies. Weed surveys indicated nearly half of the total grain growing area was moderately to heavily infested with wild oat. In situations where wild oat was the dominant weed, yield losses reportedly ranged as high as 50%.

Through the 1950's and 60's the chief cultural method of controlling wild oat was by delayed seeding. This practice entailed one or more cultivations in the spring to stimulate flushes of wild oat that could then be worked down prior to seeding. Not only did this shorten the growing season, but it contributed to significant moisture loss which, in turn, imposed an additional limit on crop yield potential[4].

Based on widespread recognition among farmers, grain companies and the Canadian Grain Commission of the exorbitant costs inflicted on the Canadian grains industry by wild oat, a Wild Oat Action Committee was struck in 1973. The Committee's principal objectives were to increase awareness of the problem and to stimulate additional research activity on wild oat. The end goal was to arrest the losses caused by wild oat, henceforth labelled "The Great Grain Robber".

In the ensuing five years, provincial subcommittees comprising representatives of the grains industry, universities, Agriculture Canada, provincial extension departments, farm press and the agricultural chemical industry concentrated their efforts on promoting the cause[7]. At the same time, Agriculture Canada diverted significant funding towards research on wild oat through a contracts grant program. From 1973 to 1978, five new selective herbicides with unprecedented activity on wild oat were introduced into the marketplace (Table 1). During this time, the area of cropland treated for wild oat increased approximately four-fold (Figure 1).

Of the products commercialized in the mid-seventies, the one that had the biggest impact on grain and oilseed production across western Canada was diclofop-methyl. Not only was diclofop-methyl highly effective over a wider range of leaf stages than its predecessor barban, but it controlled green foxtail (*Setaria viridis*), the second most economically important grass weed on the Canadian Prairies. From the late seventies through to the mid-eighties, much of the growth in the wild oat herbicide market was from increased sales of diclofop-methyl. An analysis of the contribution of technological advances to grain production during the period 1912 to 1980 at Lethbridge, Alberta, indicated that the major increases in wheat yields occurring in the late seventies were due in large part to the introduction of this chemical[1].

Table 1.   Post-emergence wild oat herbicides used commercially in western Canada

Chemical family/ Common name	Trade name	Registration Year	Major use
Carbamate			
Barban[1]	Carbyne ®	1960	wheat, barley, flax, canola
Asulam[1]	Asulox F ®	1973	flax
Bipyridylium			
difenzoquat	Avenge ®	1974	wheat, barley
Aminopropionate			
benzoylprop-ethyl[1]	Endaven ®	1973	wheat
flamprop-methyl	Mataven ®	1978	wheat
Aryloxphenoxypropionate			
diclofop-methyl	Hoegrass ®	1976	wheat, barley, flax, canola
fluazifop-butyl	Fusilade ®	1984	flax, canola
fenoxaprop-ethyl	Excel ®	1989	flax, canola
	Triumph ®	1990	wheat
quizalofop-ethyl	Assure ®	1991	flax, canola
Cyclohexanedione			
sethoxydim	Poast ®	1983	flax, canola
tralkoxydim	Achieve ®	1992	wheat, barley
clethodim	Select ®	1992	flax, canola
Imidazolinone			
imazamethabenz	Assert ®	1989	wheat, barley

[1]   No longer commercially available.

From 1982 through 1989 the area treated with wild oat herbicides remained fairly constant with between 8 to 10 million ha treated annually. This represents about 20 to 25% of the seeded area in western Canada. Much of the cereal acreage was treated with diclofop-methyl, while sethoxydim, introduced in 1983, was the popular choice for use in broadleaf crops such as flax and lentils. For the most part, these herbicides provided consistently good to excellent control of the wild oat. Infrequent performance failures usually were ascribed to incorrect application, wrong timing, or drought conditions at the time of treatment. In areas of the Prairies where there is no summer fallow and the land is cropped continuously, some fields were sprayed for wild oat every year for more than a decade.

Herbicide Resistance

Ironically, the seeds of resistance were carried in the very properties that contributed to the increased usage of the "new generation" wild oat herbicides. Because they were highly effective, often providing 95% or better control of wild oats, and because they were 'single site toxicants', these chemicals exerted intensive selection pressure. According to theoretical models, the most important parameter affecting the evolution of resistance within weed populations is selection pressure, with other factors such as seed longevity and relative fitness playing a lesser role[2].

In the fall of 1990 four farmers, three from northwestern Manitoba and one from southern Saskatchewan, reported unsatisfactory control of wild oat treated with diclofop methyl or the newly registered Triumph Plus® which contains fenoxaprop-ethyl. This herbicide, like diclofop-methyl, belongs to the aryloxyphenoxypropionate family of herbicides. A review of herbicide use on the affected fields indicated that they had been sprayed with diclofop-methyl or sethoxydim for at least eight of the past ten years[3].
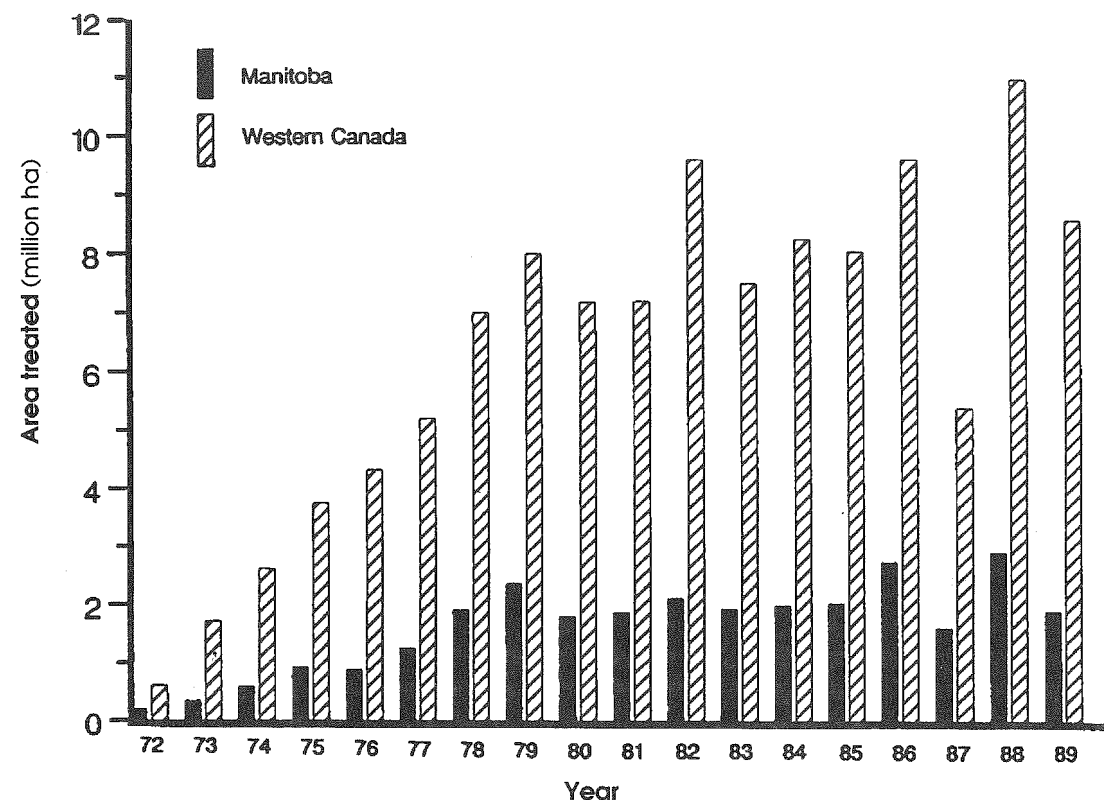
Figure 1. Area treated with wild oat herbicides in western Canada, 1972-89. Source: Economics Branch, Manitoba Agriculture.

From a dose-response trial conducted in the field it was determined that more than 8 times the recommended dosage of fenoxaprop-ethyl was required to control the resistant population (UM1). In contrast, a susceptible population, UM5, was completely controlled at the recommended dosage of 100 g ha^{-1} (Figure 2).

$$y = (k/(1+e^{bg}x^b))+d$$
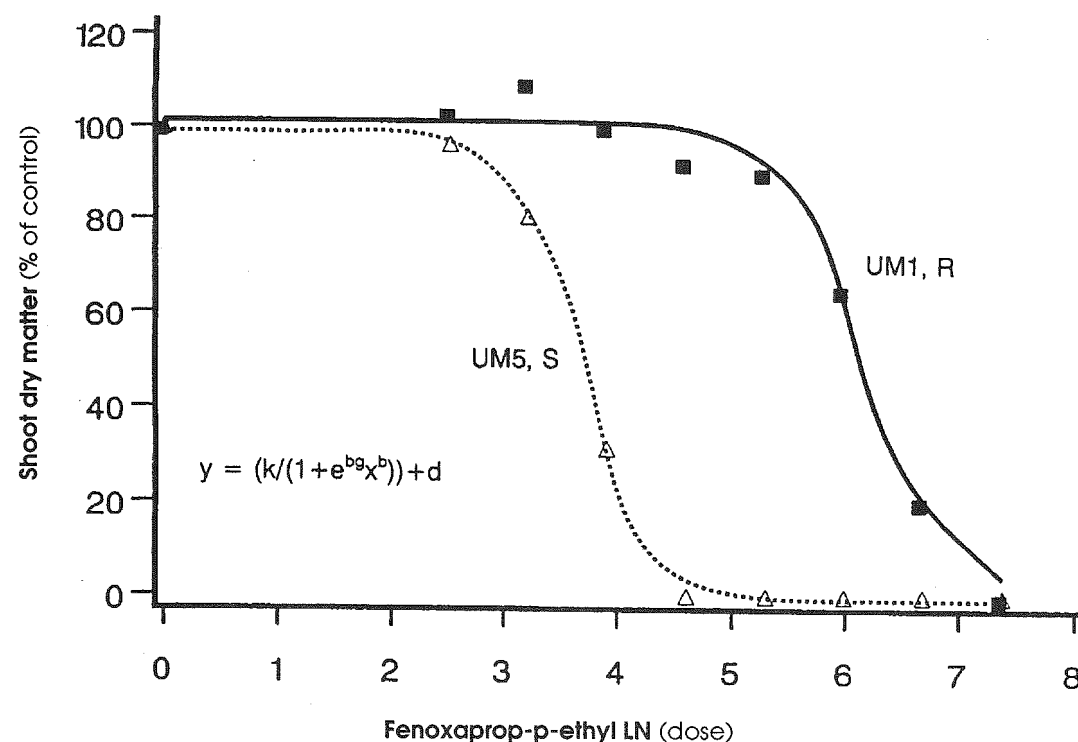
Figure 2. Response of UM1, a resistant wild oat population, and UM5, a susceptible population, treated with fenoxaprop-p-ethyl in wheat, 1991.

Comparisons of GR50's, i.e. the dosages required to reduce dry matter accumulation by 50% three weeks after treatment, indicated that the resistant populations were approximately 6 to >20 times more resistant to diclofop-methyl than a susceptible population, and from 14 to >74 times as resistant to fenoxaprop-ethyl[3]. These levels of resistance are many times higher than previously reported for either tame or wild oat[5,10].

Population UM1 also proved to be extremely resistant to sethoxydim with an R/S ratio >150, whereas the other two populations from Manitoba (UM2 and UM3) showed intermediate resistance with R/S ratios of under 10. The population from Saskatchewan (UM33) was unique in that it was highly resistant to both diclofop-methyl (R/S >>20) and fenoxaprop-ethyl (R/S >74), but not to sethoxydim.

Whereas, diclofop-methyl and fenoxaprop-ethyl are aryloxyphenoxypropionate herbicides and have chemical structures distinctly different from sethoxydim which is a cyclohexane-dione, both classes of herbicides interfere with acetyl-CoA carboxylase (ACCase) in susceptible plants[8]. Hence it might well be expected that any change that confers resistance to one class might also confer resistance to the other. However, the fact that UM33 was resistant to the aryloxyphenoxypropionates, but not to sethoxydim, indicates that this relationship is not absolute.

The differences in levels and patterns of cross resistance among populations indicates that resistance has arisen from separate, distinctly different mutations. While the precise mechanism(s) of resistance has not been determined, physiological and biochemical investigations with UM1 indicate that resistance is not associated with altered uptake, translocation or metabolism of the herbicides. Neither does it appear to be directly linked to differences in ACCase sensitivity to the herbicides (see Devine, these Proceedings). Whatever the precise mechanism of resistance, initial indications from genetic studies indicate that the trait is controlled by a single, semi-dominant, nuclear gene (Murray, unpublished).

Since resistance was first confirmed in the fall of 1990, more than sixty additional wild oat populations have been confirmed to be resistant to the ACCase inhibitors. As well, O'Donovan et al.[9] have identified resistance to the soil-applied herbicide triallate in approximately twenty wild oat populations from Alberta. These populations are insensitive to triallate at up to four times the recommended rate, and peculiarly are also cross-resistant to the foliar-applied herbicide, difenzoquat at up to two times the recommended dosage. The resistant plants were selected from fields that had been continuously treated with triallate for between 10 and 15 years.

Among the ACCase inhibitor resistant populations tested to date, there is no evidence of cross-resistance to herbicides including difenzoquat, imazamathabenz or flamprop-methyl which belong to different chemical families and have different modes of action. In the short-term these herbicides could be used to combat resistant wild oats or used in a herbicide rotation to delay or avoid the occurrence of resistance.

A Look to the Future

Projecting to the future, it is inevitable that resistance is going to become an increasingly serious problem. Since its introduction in 1990, fenoxaprop-ethyl has rapidly become a preferred herbicide in wheat, partly due to its superior activity on wild oat and green foxtail and partly because of its compatibility with thifensulfuron a broad-spectrum, broadleaf weed herbicide also used in that crop. With the exception of imazamethabenz, all products registered for wild oat control since 1990 are ACCase inhibitors, including tralkoxydim which was introduced in 1992 and is expected to take a significant share of the wild oat market. The advantage of this product is that it not only controls wild oat and green foxtail, but it can be applied to both barley and wheat and can be tank-mixed with bromoxynil/MCPA for broadleaf weed control.

While provincial extension efforts have created awareness of the resistance problem among farmers, a relatively small minority are consciously planning their weed control program to avoid the problem. The majority are either continuing to use the same herbicides that have worked well for them in the past or are shifting to other ACCase

inhibitors. The immediate task is to convince farmers of the need to rotate herbicides among products with different modes of action as a resistance avoidance strategy. In the long term, the problem may well demand reduced reliance on herbicides to control wild oat which has become accepted practice for the past fifteen to twenty years.

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The Biochemical Basis of Herbicide Resistance in Wild Oat

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Summary

The mechanisms of herbicide resistance in several wild oat (*Avena fatua* and *A. sterilis*) populations have been examined in recent years. In Canadian wild oat biotypes resistant to diclofop-methyl, resistance cannot be ascribed to differences in herbicide uptake, translocation, metabolism to inactive products, or sensitivity of the target enzyme (acetyl-coA carboxylase; ACCase) to the herbicide. In one biotype, resistance has been correlated with recovery of the plasma membrane electrogenic potential after herbicide treatment. In one wild oat biotype from Australia, resistance has been associated with an altered form of ACCase with reduced herbicide sensitivity. No information is available on the resistance mechanism in wild oat biotypes resistant to thiocarbamate herbicides or difenzoquat.

Introduction

Wild oat resistant to several classes of herbicides have been reported in recent years, primarily in western Canada and in Australia[7,9,11]. Various aspects of the herbicide resistance and cross-resistance of these populations have been characterised, and the results are presented elsewhere (*see also* papers by Morrison and Holtum, this volume). In most instances, resistance has arisen following repeated selection with one herbicide or with herbicides from the same chemical family or having the same mechanism of action.

Several possible mechanisms can be suggested to explain herbicide resistance in weeds. The obvious mechansims include reduced herbicide interception and retention, reduced uptake and translocation, altered herbicide metabolism, resulting in smaller pools of active herbicide available in the tissue, and reduced sensitivity of the target site to the herbicide. In addition, several other possible resistance mechanisms, which will be discussed later, can be considered in special cases.

In this paper the resistance mechanisms of several herbicide-resistant wild oat biotypes from western Canada and Australia are discussed. All of the Canadian wild oat biotypes are *A. fatua*, whereas the Australian populations include both *A. fatua* and *A. sterilis*.

Mechanism of Resistance to ACCase Inhibitors

In wild oat biotypes that were identified in the early 1980s as being diclofop-methyl resistant, resistance was not associated with differences in foliar interception or retention of herbicide spray[9]. Both the resistant and susceptible biotypes were initially injured by diclofop-methyl, but the resistant biotypes recovered within 7 to 9 days after treatment. In subsequent research, it was shown that uptake of diclofop-methyl and translocation from the treated leaf to other plant parts was equal in the resistant and susceptible biotypes[4].

Tolerant crops such as wheat rapidly metabolize diclofop-methyl to the parent free acid (diclofop), which is subsequently glycosylated to inactive products[8,16]. The aryl glycosides formed are considered terminal metabolites. Susceptible species such as wild oat and cultivated oat de-esterify the diclofop-methyl to diclofop, but convert the free acid principally to glycoside esters rather than aryl glycosides. The glycoside esters represent temporary metabolites from which diclofop can be regenerated by de-esterfication[15].

Research on diclofop-methyl metabolism in the earlier-identified resistant and susceptible wild oat biotypes showed no differences in the rate or degree of metabolism of diclofopmethyl to diclofop and subsequently to more polar conjugates[4].

In addition, the relative amounts of aryl glycosides of diclofop and glycosyl esters in the two biotypes did not differ. Thus differential metabolism was ruled out as a possible mechanism of resistance.

Diclofop and related herbicides inhibit the enzyme acetyl-coA carboxylase (ACCase), a key enzyme in fatty acid biosynthesis[10]. Resistance in broadleaf species to herbicides such as diclofop is based on the insensitivity of ACCase from these plants to the herbicides. Consequently, it might be expected that resistance in the wild oat populations would be based on a similar alteration in the target enzyme. This is indeed the case with a resistant wild oat biotype from Australia (S.B. Powles, pers. comm., 1992); in this biotype resistance at the whole plant level is correlated with *in vitro* insensitivity of the target enzyme. However, this particular biotype may be an exception rather than the rule in terms of mechanisms of resistance to ACCase inhibitors.

Partially purified ACCase from the resistant and susceptible Canadian wild oat lines referred to earlier was equally sensitive to diclofop and tralkoxydim, a cyclohexanedione ACCase inhibitor[4]. Therefore resistance cannot be attributed to an altered form of ACCase that is less sensitive to the herbicides. Overall, these results are very similar to those reported for a second wild oat biotype from Australia. In this biotype, no differences were found in the 150 values for ACCase from the resistant and susceptible biotypes when assayed with diclofop[1]. In addition, the specific activities and substrate affinities of the ACCase from the resistant and susceptible biotypes were similar, suggesting no inherent kinetic differences in ACCase from the two biotypes.

More recently, similar research has been conducted on the highly resistant wild oat biotypes from Manitoba, Canada. No differences have been reported in herbicide uptake, translocation or metabolism between the resistant and susceptible biotypes. ACCase from the resistant and susceptible biotypes is equally sensitive to diclofop (and other ACCase inhibitors), again suggesting that resistance is not based on an altered target enzyme[3]. Similar results have been published on herbicide-resistant and susceptible *Lolium rigidum* biotypes from Australia[12].

Previous research has shown that diclofop has an independent effect on certain properties of the cell membrane and tonoplast, unrelated to inhibition of ACCase. The electrogenic potential of the membranes is rapidly depolarised by diclofop, and diclofop also prevents auxin-induced hyperpolarisation of the plasma membrane[13,18]. This effect can be measured in several ways, including: a) measuring the electrogenic potential (Em) of the membrane directly by inserting a microelectrode inside the cell; and b) by monitoring the effect of diclofop on the pH of an unbuffered solution containing plant tissue.

We have used both of these approaches to study the effect of diclofop on the membrane properties of resistant and susceptible wild oat tissue. To date, this constitutes the only research on this topic that has indicated any differences between the resistant and susceptible biotypes from Canada.

In normal tissues, protons (H^+) are pumped out of the cells by an H^+-ATPase located in the plasma membrane. This results in net acidification of the external solution, at least over the short term (e.g., 1 h). In susceptible tissue, acidification is reversed as soon as diclofop is added to the bathing solution, and the tissue permanently loses the ability to generate or maintain the transmembrane proton gradient[3]. In resistant tissue, however, the effect is temporary; acidification is reversed when diclofop is added to the tissue, but is restored when the diclofop is removed. Thus, although diclofop does affect the tissue's ability to generate a proton gradient, the effect is only temporary.

The corresponding result is obtained in electrophysiology experiments. The membrane of both the resistant and susceptible wild oat is depolarised when diclofop is added to the tissue, but is restored in the resistant biotype when the diclofop is removed from the bathing solution[3]. Again, a similar result has been reported with herbicide-resistant and susceptible biotypes of *Lolium rigidum* from Australia[6].

Collectively, these results suggest that diclofop can affect membrane function in both resistant and susceptible biotypes, but that the resistant biotypes are able to recover. While this correlates well with resistance at the whole-plant level, it does not explain it or provide a mechanism of resistance.

There is some controversy concerning the relevance of the effect of diclofop on membrane properties to its mechanism of action. While some researchers have argued this biophysical action is an important component of its total phytotoxic action, others have concluded that it is irrelevant to the herbicial action of diclofop, and that ACCase is the only important target site[e.g.,5,14]. This is not a simple question to resolve, and further research, perhaps with these apparent "membrane mutants," is required before the contribution of the membrane effect to the overall action of these herbicides can be determined.

Speculation on possible mechanisms

The mechanism by which diclofop affects the electrogenic properties of the cell membrane is not well understood. However, diclofop does not act simply like a proton ionophore such as CCCP[14]. Membrane depolarisation by diclofop can be prevented by PCMBS, a non-permeant molecule that binds -SH groups on proteins[14]. This suggests that diclofop interacts with a protein in the membrane, or closely associated with the membrane, in such a way as to allow protons to flow back into the cell. Recently we have used purified plasma membrane vesicles to study the effects of diclofop on various properties of the cell membranes from resistant and susceptible wild oat. We have found no differences in the effect of diclofop on the plasmalemma H^+-ATPase (the major component of the membrane contributing to the proton gradient) in resistant and susceptible wild oat, or on the electrochemical gradient (Renault and Devine, unpublished results). However, it is possible that the effect of diclofop on the cell membrane, and the basis for the differential sensitivities of the resistant and susceptible wild oat, is mediated through an effect on a protein peripheral to the membrane. If this were the case, experiments with plasma membrane vesicles would not necessarily identify the important difference between the two biotypes.

The paradoxical finding in this research is that ACCase from both the resistant and susceptible wild oat is sensitive to diclofop when assayed *in vitro*, but the resistant wild oat is not affected by the herbicide *in vivo*. A possible alternative explanation for resistance is that the resistant enzyme is modified during the extraction and assay procedure, such that it "appears" to be sensitive. However, this is unlikely, since in other species (e.g., *Setaria viridis*), resistance to ACCase inhibitors is based on an altered form of ACCase, and the different sensitivities of resistant and susceptible ACCase is seen clearly in the in vitro enzyme assays (Marles and Devine, unpublished results).

A second possibility is that the resistant wild oat biotype is a "sequestration mutant," i.e., it possesses some mechanism for preventing the herbicide from gaining access to the ACCase in the plastid. Somehow, this mechanism must be able to discriminate between herbicides that are ACCase inhibitors and other herbicides. Although sequestration has been proposed as the mechanism of resistance to bipyridilium herbicides in some species, there is as yet no evidence that it is involved in wild oat resistance to ACCase inhibitors.

Resistance to Other Herbicides

Wild oat biotypes resistant to the thiocarbamate herbicide triallate were identified in western Canada in the late 1970s[17]. Interestingly, some of these biotypes showed some cross-resistance to diclofop-methyl. Since these two herbicides are believed to have quite different modes of action, it is unlikely that resistance is due to a target site mutation. However, no research has been reported on possible mechanisms of resistance in these biotypes.

Triallate-resistant wild oat biotypes have been identified recently in Alberta, Canada. Apparently, these biotypes are cross-resistant to difenzoquat, but not to other herbicides. This finding raises some interesting questions about the mechanism of action of difenzoquat; although several research papers have been published on various aspects of difenzoquat action in plants, no definitive target site has been identified.

Triallate and related herbicides inhibit the elongation of fatty acids in plants, by interfering with the "elongase" system[2]. If resistance to triallate and cross-resistance to difenzoquat in these wild oat populations is based on a target site mutation, this would provide direct

evidence for a target site for difenzoquat. However, this possibility has not yet been examined.

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Herbicide Resistant Wild Oats in Australia

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Summary

Herbicide resistance of two species of wild oats (*Avena fatua* and *Avena sterilis* ssp *ludoviciana*) to the aryloxyphenoxypropionate and the cyclohexanedione herbicides in Australia has been confirmed. So far, one biotype is exhibiting a herbicide-insensitive acetyl-coenzyme A carboxylase which is conferring resistance to the above herbicides. Surprisingly, the number of herbicide applications to which the resistant biotypes have been exposed varied, ranging from less than five years to over ten. Farmers must therefore adopt integrated weed management strategies if they are to avoid the problem.

Introduction

Herbicide resistance was first documented in 1990 for a biotype of *Avena fatua* from near York in Western Australia[1,6]. By August 1992, resistance to the aryloxyphenoxypropionate (the so-called '-fops') herbicides and, to a lesser extent to the cyclohexanediones (the so-called '-dims') had been reported in approximately 20 biotypes from Western Australia (Gill unpub.), South Australia and New South Wales (Holtum, unpub.). Resistance has been reported in both of the wild oat species that infest cropping lands in Australia viz. *Avena fatua* and *Avena sterilis* ssp *ludoviciana*. In Australia, no resistance has been reported to herbicides other than the aryloxyphenoxypropionates and cyclohexanediones.

Patterns of Resistance

Two broad patterns of graminicide resistance have been documented in Australian wild oat biotypes. Some exhibit resistance only to diclofop. In these populations the level of resistance is low, typically 2 to 3 fold, but sufficient to be agronomically relevant[6]. Other populations exhibit high resistance to all '-fop's (both selective and non-selective) and low resistance to '-dim's[5].

Resistant wild oats in western Canada (see Morrison *et al* and Devine this volume) exhibit these patterns and others. The patterns not yet observed in Australia include high resistance to '-fop's and '-dim's or high resistance to '-dim's but low resistance to '-fop's. It can be expected that the patterns not yet documented in Australia may manifest themselves as the resistance phenomenon becomes more widespread. During the late 70's and early 80's there were rare occurrences of resistance to triallate and flamprop-methyl in *A. fatua* in North America. No such populations were described in Australia.

There is no evidence to suggest that the patterns of resistance differ between *A. fatua* and *A. sterilis*.

Mechanisms of Resistance

Two mechanisms of resistance to the '-fop' and '-dim' herbicides have been detected in wild oats. Some biotypes possess herbicide-insensitive acetyl coenzyme A carboxylase (ACCase), the target enzyme of the '-fop' and '-dim' herbicides[4]. Other populations contain herbicide-sensitive ACCase but possess a mechanism that is characterised by an ability to repolarise membranes following exposure to graminicides (Devine this volume). This latter mechanism, which still awaits a physiological explanation, has been reported for some biotypes of herbicide-resistant *Lolium rigidum* (annual ryegrass)[2,3].

Herbicide-insensitive ACCase has been extracted from three Australian wild oat populations. In the best characterised population, *A. sterilis* biotype SAS 1, resistance at the whole plant level corresponds reasonably well with the levels of resistance of the ACCase *in vitro* (Table 1).

Table 1. Resistance to herbicides in *A. sterilis*, biotype SAS 1, compared with that of a susceptible *A. sterilis*, biotype SAS 2. Resistance is expressed as a ratio of the rates of herbicide required to kill 50% of plants or required to inhibit ACCase by 50 %.

Herbicide	Resistance of whole plants (ratio of LD50 values)	Resistance of ACCase (ratio of Km values)
diclofop	>213	52
fluazifop	>1067	9
haloxyfop	181	25
tralkoxydim	2.7	6
sethoxydim	2.5	8

The mechanism of resistance in a diclofop-only resistant population of *A. fatua*, WAF 1, has not been resolved. Although an insensitive ACCase was not detected nor were differences in uptake and metabolism of 14C-labelled diclofop observed[1], it is not clear what differences in ACCase sensitivity might be necessary to produce the only 2 to 3 fold resistance observed at the whole plant level.

Practices that have Resulted in Resistance

Little is known of the importance of farm management systems to the development of resistance in wild oats in Australia. Some populations have been exposed to more than ten years of '-fops' and exhibit little or no resistance whereas others have developed resistance after considerably less exposure. This is demonstrated in Table 2 which gives the paddock histories for WAF 1, a moderately diclofop-only resistant *A. fatua* biotype from Western Australia and biotype SAS 1, a highly '-fop' resistant *A. sterilis* biotype from South Australia.

Table 2. Herbicide histories, supplied by growers, for fields from which herbicide-resistant *A. fatua*, biotype WAF 1, and *A. sterilis*, biotype SAS 1, were collected.

Year	Crop Sequence WAF 1	Herbicide WAF 1	Crop Sequence SAS 1	Herbicide SAS 1
1979	wheat	diclofop	—	—
1980	lupins	diclofop	—	—
1981	wheat	diclofop	wheat	diclofop
1982	lupins	diclofop	pasture	none
1983	wheat	diclofop	none	trifluralin, fluazifop
1984	lupins	diclofop	rape seed	trifluralin, fluazifop
1985	wheat	diclofop	wheat	triallate, diclofop
1986	—	—	wheat	diclofop
1987	—	—	clover	trifluralin, fluazifop
1988	—	—	wheat	glyphosate
1989	—	—	clover	trifluralin, haloxyfop

In all probability resistance in wild oats has been slower to manifest itself than in ryegrass (*Lolium rigidum*) because wild oat populations are generally less dense than ryegrass and the species are not as variable as ryegrass. Other factors such as a self-pollinating habit and a suspected slower seed bank turnover time may also play roles. The phenomenon is too recent for such studies to have been made.

Some fields with herbicide resistant wild oats also contain herbicide resistant ryegrass but many do not. In one study where resistant wild oats (*Avena sterilis* ssp *ludoviciana*) and ryegrass were collected from the same paddock, the patterns of resistance were broadly similar. Both species were resistant to the '-fops' and '-dims' and resistance was greater to the '-fops' (Table 3). The physiological bases for resistance in the two species have not been studied.

Table 3. A comparison of resistance of wild oats and annual ryegrass collected from a single field. Values are the ratios of rates of herbicides required to kill 50 % of susceptible populations. (n.d. = not determined.)

Herbicide	Annual ryegrass	Wild oats
diclofop	>30	>23
fluazifop	>45	28
haloxyfop	>16	11
quizalofop	n.d.	12
fenoxaprop	n.d.	>16
tralkoxydim	5	0.5
sethoxydim	5	2
chlorsulfuron	1.5	n.d.

The Future

Experience in both Australia and North America suggests that the continued widespread reliance on selective herbicides such as the '-fops' and '-dims' for the control of large populations of wild oats will select for more resistance. The extent, severity and, ultimately, the economic impact of resistance will depend upon the rapidity with which growers adopt integrated weed management practises that exhibit non- or low- selectivity for resistance.

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New Sources of Herbicide Resistance in *Avena Spp.*

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Summary

In 1990 and 1991, over 8,000 accession lines from the USDA World Oat Collection were screened for resistance to seven wild oat herbicides including imazamethabenz, triallate, difenzoquat, fenoxapropethyl, diclofop-methyl, flamprop-methyl, and sethoxydim. A total of 142 lines belonging to six *Avena* species (viz. *A. sativa, A. brevis, A. byzantina, A. nuda, A. abyssinica* and *A. strigosa*) were found to have some resistance to wild oat herbicides. Asia Minor and southeastern Europe appear to be the main gene centres for herbicide resistance, and Spain, Portugal and Ethiopia as possible other centres of resistant germplasm.

Introduction

Oat (*Avena sativa* L.) shares with wheat and barley the distinction of being one of the most important cereal crops in western Canada. Statistical abstracts show large annual fluctuations in hectareage, production and value, but roughly 1.5 million hectares are used each year to produce about 2.5 million metric tonnes of oat with an annual value of approximately $395 million[5].

Although the older cultivars of oat grown in Canada consisted of low yielding and agronomically inferior genotypes, oat breeders have in recent years developed new cultivars that are high yielding and well adapted to the soils and climates of western Canada. However, these yield improvements have eluded many farmers because insufficient attention has been directed towards developing methods of controlling wild oat (*Avena fatua* L.).

Wild oat causes greater yield loss than any other weed species and infests approximately 85% of the cultivated land in western Canada[1]. Friesen and Shebeski[4] estimated crop yield reductions ranging from 5 to 50% in fields in which wild oat was the dominant weed species. Although acceptable chemical control of wild oat has been obtained in wheat and barley, a similar level of wild oat control in oat has not been possible because of a lack of suitable herbicides that can control wild oat without injuring cultivated oat. This problem could have been solved if new herbicides were developed or new cultivars with resistance to existing herbicides were bred. Research in these two areas has not been strong. Because of stringent regulations pertaining to toxicologic data, and increasing costs of research and development, herbicide manufacturers have not had the economic incentive to develop new herbicides. On the other hand, oat breeders were unable to develop herbicide resistant cultivars because genetic sources of herbicide resistance are unavailable.

There is a tremendous amount of genetic diversity in oat[3] and a large collection of strains and cultivars is assembled in world oat collections. To locate the desired genes, the obvious place to turn is to these germplasm collections, which have not been previously screened for herbicide resistance. This study was conducted to search for new genes that can confer resistance to wild oat herbicides. For practical reasons, our specific objective was to identify new sources of herbicide resistance that are conditioned by either cytoplasmic genes or nuclear-cytoplasmic genetic systems.

Materials and Methods

The experiment was carried out at Agriculture Canada, Lacombe Research Station, in the summer of 1990 and 1991. During the two years, a total of 8042 accession lines from the USDA World Oat Collection were screened for resistance to several wild oat herbicides.

These accession lines represented six *Avena* species collected from all known centres of origin, diversification and domestication of oat. Each accession line was screened for seven (one pre-emergence and six post-emergence) herbicides. The pre-emergence herbicide, Avadex BW® (triallate), was applied at the rate of 1.7 kg ha^{-1} and the soil incorporated twice before planting. Only 3072 accession lines that were grown in 1990 were screened for resistance to this herbicide. The post-emergence herbicides Assert® (imazamethabenz), Hoegrass® (diclofop-methyl), Poast® (sethoxydim), Avenge® (difenzoquat), Excel® (fenoxaprop-ethyl) and Mataven® (flamprop-methyl) were applied at rates of 0.50, 0.80, 0.30, 0.83, 0.20 and 0.26 kg ha^{-1}, respectively, when the majority of the seedlings were in the 2 to 4 leaf stage. All 8042 accessions were screened for resistance to these six post-emergence herbicides. All post-emergence herbicides were applied in 100 L ha^{-1} of water at 275 kPa using a motorized plot sprayer with TeeJet® 8001 flat-fan nozzles. Two to three weeks after spraying, the accession lines were scored for herbicide resistance using a zero (no resistance) to nine (very resistant) scale.

Results and Discussion

From among the 8042 accessions that were screened in 1990 and 1991, 142 lines belonging to six *Avena* species (viz. *A. sativa, A. byzantina, A. strigosa, A. brevis, A. nuda*, and *A. abyssinica*) were found to have some resistance to wild oat herbicides. Of these 142 lines, 91, 38, 8, 4, and 1 accessions were resistant to diclofop-methyl, flamprop-methyl, fenoxaprop-ethyl, difenzoquat and imazamethabenz respectively. About 20% of the herbicide resistant accessions showed multiple resistance with the majority showing concurrent resistance to diclofop-methyl and flamprop-methyl. Some of the accession lines showed resistance to more than one herbicide.

More herbicide resistant lines were found in *A. sativa* than in any of the other *Avena* species. Out of the 142 herbicide resistant lines, 84 were *A. sativa*, whereas less than 10 herbicide resistant lines were identified in each of the other five species. Compared to the other species, *A. sativa* also showed a wider spectrum of herbicide resistance including resistance to such herbicides as diclofop-methyl, flamprop-methyl, difenzoquat and fenoxaprop-ethyl.

More accession lines showed resistance to diclofop-methyl than to the other six herbicides, and lines resistant to this herbicide were identified in all species with the exception of *A. abyssinica* (Table 1). Some of the diclofop-methyl resistant lines appeared to have a higher level of diclofop-methyl resistance than Savena I[2] and Saia-4[6]. Although the genetic systems controlling resistance in each of the 91 diclofop-methyl resistant lines are not yet known, we speculate that the herbicide resistance genes that we discovered in *A. nuda, A. brevis, A. fatua* and *A. byzantina* may be different from the diclofop-methyl resistance genes that were previously discovered in *A. strigosa*[2] and *A. sativa*[6].

The existence of genes that confer resistance to flamprop-methyl has not been previously reported in the scientific literature. In the present study, 38 accessions showing resistance to flamprop-methyl were identified in four species including *A. abyssinica, A. brevis, A. sativa* and *A. strigosa*. (Table 1). Out of the 8032, only four lines with resistance to difenzoquat were identified, and all four were *A. sativa* lines. In addition, eight accession lines (three *A. sativa*, one *A. brevis*, one *A. nuda* and three yet unclassified lines) showed resistance to fenoxaprop-ethyl, but the level of resistance expressed in these lines was relatively low. None of the 8042 lines that were screened in 1990 and 1991 showed resistance to sethoxydim. and none of the 3072 lines that were screened in 1990 were resistant to triallate. Only one accession line with resistance to imazamethabenz was identified, but its taxonomic classification is not yet established (Table 1).

The six *Avena* species that we examined in this study have evolved in different parts of the world and at different times on the evolutionary time-scale. Therefore. we postulate that several genes or gene combinations may confer resistance to wild oat herbicides. Asia Minor and southeastern Europe appear to be the main gene centres for herbicide resistance, and Spain, Portugal and Ethiopia are other possible centres of resistant germplasm. As far as we know, the existence of genes that confer resistance to flamprop-methyl, imazamethabenz, fenoxaprop-ethyl and difenzoquat has not been previously reported in the scientific literature.

This report therefore represents the first paper showing the existence of genes that may confer resistance to the aforementioned herbicides.

Table 2. Number of herbicide resistant lines by species and herbicides[1].

Species	Imazametha-benz	Difenzo quat	Diclofop-methyl	Flamprop-methyl	Flamprop-ethyl	Total
A. abyssinica	-	-	-	2	-	2
A. brevis	-	-	5	3	1	9
A. byzantina	-	-	7	-	-	7
A. fatua	-	-	1	-	-	1
A. nuda	-	-	1	-	-	2
A. sativa	-	4	60	17	3	84
A. strigosa	-	-	4	3	-	7
Unknown	1	-	13	13	3	30
Total	1	4	91	38	8	142

[1] No accession lines with resistance to sethoxydim or triallate were identified.

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Breeding for Herbicide Resistance in Oats: Opportunities and Risks

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Summary

Genetic variation for herbicide resistance is available within the oat genome and it is possible to breed herbicide resistant varieties. If wild oats share the same agricultural ecosystem with the herbicide resistant varieties, interspecific hybrids will occur and herbicide resistant wild types will evolve and render the recommended herbicide/s ineffective. The agricultural impact of herbicide resistant varieties will therefore hinge on the management of the oat crop, the frequency of outcrossing, the fitness of the interspecific hybrids, the management of the F1 and subsequent generations.

Introduction

Grass weeds such as *Lolium, Bromus, Vulpia, Hordeum, Setaria, Alopercurus* and particularly *Avena* are often major competitors reducing the yield and, sometimes, quality of oat crops. With few graminicides registered, and few likely to become available, the selective control of most of these weeds is likely to remain difficult in oat crops. Only chlorfenprop-methyl has been registered for control of *A. fatua* in some oat varieties[18]. There is little current research on two alternative strategies; herbicide antidotes (or safeners) or allelopathy.

There are at least seven reports of genetic resistance to graminicides identified in wild or cultivated oats and the transfer of these into acceptable varieties would increase the options for grass weed control. In this paper, opportunities for breeding herbicide resistant varieties and the risk of herbicide resistance genes introgressing into the weedy species is assessed by considering: 1. the characteristics of the genotype to accept the herbicide resistance gene/s; 2. the likelihood of hybridisation and survival of subsequent generations, and; 3. management strategies for the herbicide resistant variety.

Sources of Resistance to Graminicides

In this review, only resistance in *Avena* to herbicides which act primarily on the enzyme acetyl CoA carboxylase, active on all three major wild oat species (*A. fatua, A. sterilis* and *A. barbata*) plus other grass weeds (Table 1). are considered.

Widespread applications of the first aryloxyphenoxypropionate (AOPP) herbicide, diclofop-methyl, registered by Hoeschst in 1978 has imposed intense selection pressure favouring resistant individuals in wild oat populations. Other AOPP and cyclohexanedione (CHD) herbicides have added to this pressure and resistance has been reported in at least Australia and Canada in both *Avena fatua* and *Avena sterilis*[16,11,15].

Resistance in cultivated oats to the AOPP herbicide diclofop-methyl was first reported by Taylor and Codd[17] and Barr[1] (Table 1). Barr[1] also demonstrated resistance in *Avena strigosa* and a diclofop-methyl resistant *Avena sativa* germplasm "Savena 1" was registered in 1987[2]. Warkentin *et al.*[20] tested nearly 350 genotypes, mainly from North America, for tolerance to diclofop-methyl but found none as resistant as Savena 1. Kibite and Harker[10] systematically tested the resistance to graminicides of 8042 accessions of *Avena* species from all three ploidy levels. Details of the herbicide spectrum and genetic control of these and other resistances are listed in Table 1.

For breeding, the most promising source of resistance is SAS1 as it confers resistance to many herbicides, facilitating selective control of a wide range of weedy grasses. It also confers a wide safety margin as the SAS1 biotype tolerates applications 10 times the recommended dose rate necessary for control of most grassy weeds[11].

In contrast, transfer of resistance to a single herbicide, such as from the York or Savena 1 genotypes (Table 1), has the advantage that any "escape" could then be controlled by other AOPP herbicides as well as CHD's, phenylureas and carbamates. Unfortunately, the margin between application rates for control of the target weed and crop tolerance is narrow with these mono-herbicide resistant genotypes.

Table 1. The genotypes, spectrum and genetic control of herbicide resistances reported in *Avena* spp.

Genotype(s)	Species	Spectrum of Resistance Reported	Genetics of Resistance	Ref.
SAS1	*A. sterilis*	most AOPP	Single incompletely dominant gene	4,11
York	*A. fatua*	diclofop-methyl only	Unknown	16
Saia	*A. strigosa*	diclofop-methyl	Unknown	1
Savena 1 (Algerian, NZ Cape)	*A. sativa*	diclofop-methyl	Two recessive genes	2,20
Elen	*A. sativa*	diclofop-methyl	Not reported	19
UM 1	*A. fatua*	AOPP and CHD	Not reported	15
148 lines	*A. sativa, A. brevis, A. byzantina, A. nuda, A. strigosa, A. abysinnica*	7 different herbicides	Not reported	10

Breeding Herbicide Resistant Oat Varieties

Incorporation of the AOPP-resistance gene[4] from SAS1 into cultivated oats is amenable to routine backcrossing procedures. Homozygous, dominant, resistant types are being distinguished from the heterozygous, partially resistant types, by judicious selection of herbicide rates for screening[4]. A transfer program in South Australia is currently at BC_2F_2 and top $cross_2F_2$. No linkage with undesirable traits nor pleiotropic effects of the resistance gene have been detected so cultivars could be available in 1997. The two recessive genes of Savena 1 should also be amenable to incorporation by routine backcrossing to improved genotypes.

If cultivated oat genotypes with herbicide resistance are to be successful, the risk of outcrossing with wild species must be minimised. Hence, cleistogamous (low "promiscuity") genotypes susceptible to graminicides other than the AOPP's should be sought. Selection for genotypes with synchronous tillering, very low propensity to shattering, low post-harvest dormancy, large seeded and awnless would also help minimise the proportion of progeny from interspecific hybrids emanating from crosses between wild and cultivated oat species which may be competent as weeds.

Risk of Introgression of Herbicide Resistance into Wild *Avena*

Outcrossing

Avena sativa, A. fatua and *A. sterilis* are all hexaploid, interfertile members of the same biological species and hybrids occur occasionally in the field. The frequency of outcrossing between wild and cultivated hexaploids has been reported as 0.1 to 0.31%[9] and 0.65%[8]. Bickelman and Leist[5] showed a range in the frequency of outcrossing between varieties of

hexaploid oats of 0.5% to 4.7%. Varieties also differed in their ability to pollinate emasculated florets in the field, inferring that crossing in both directions (wild x cultivated and cultivated x wild) will be affected by the genotype of the cultivated oat.

Survival of the F_1

For most traits conferring "weediness", the F_1 will resemble the cultivated parent more closely (Table 2). It will be non-dormant, non-shattering, non-articulate and not strongly awned, semi-dwarf, large seeded and with synchronous tiller development. The F_1, from SAS1 but not Savena 1, will possess sufficient resistance to survive application of AOPP herbicides but not CHD, carbamate, phenylurea, dinitroaniline herbicides nor non-selective herbicides such as glyphosate or the bipyridyls.

Table 2. Inheritance of characters which influence the competitive ability and survival of hybrids between wild and cultivated oats

Character	Survival Value	Inheritance	F_1 Status	F_2 Ratio	Ref.
Shattering	Seed dispersal	Single recessive	Non-shattering	3 Non: 1 Shattering	18
Dormancy	Spread germination within and between seasons	3 genes, dose dependent, germination dominant	Non-dormant		9
		Single recessive	Non-dormant	3 Non-Dormant: 1 Dormant	18
Articulation	Seed dispersal	Single recessive	Non-articulate	3 Non-Articulate: 1 Articulate	9
Asynchronous tillering	Ensure escape from harvesting	Unknown	Resembles cultivated type	Unknown	
Awns	Seed burial	Single recessive	Awnless	3 Awnless: 1 Awned	9
Height	Competitive ability	Single dominant	Dwarf	3 Dwarf: 1 Tall	7

Survival of the F_2

The frequency of segregants carrying all of the weedy traits (shattering, dormancy, small seed, articulate, strong awns, high tillering, tall, asynchronous tiller development) plus herbicide resistance is estimated to be $3/2^{14}$ (Table 2). However, the frequency of dormant, shattering, awned types with herbicide resistance is $3/2^8$ and this may more closely estimate the frequency of weedy segregants. The corresponding frequencies for the Savena 1 genes are $1/2^{15}$ and $1/2^9$ respectively.

Evaluating the Risks

The landrace Algerian was introduced into Australia in the late 1880's and it dominated the Australian oat industry for over seventy years[3]. Algerian, NZ Cape (a selection from Algerian) and other varieties in this group are all tolerant of diclofop-methyl[1]. Hence, if there were a risk of introgression of the herbicide resistance gene into wild populations, one would have expected the release of Hoegrass® (diclofop-methyl) in 1978, to select rapidly for the predominance of resistant weedy oats. Although Barr[1] reported no wild oats resistant to diclofop-methyl in 1986, there were several populations detected by 1990[17].

The emergence of SAS1 is not related to cultivated oats as its resistance is dissimilar in both the level and mode of inheritance to that in the Algerian family. The relationship between genes from cultivated oats and the other Australian herbicide resistant wild oats reported is unknown. Warkentin et. al. [20] reported that most North American varieties are susceptible to diclofop-methyl and therefore are unlikely sources for the herbicide resistance reported in A. fatua in Canada by Morrison et al. [15].

It can be argued that resistance in annual ryegrass (Lolium rigidum) and wild oats is spreading so quickly on Australian farms that the AOPP and CHD herbicides have a limited lifespan. By the time an AOPP-resistant oat variety was released, these herbicides could be declining in usage. Hence, introgression of resistance into wild types would be irrelevant. Needless to say, this is not a view which appeals to the manufacturers of AOPP and CHD herbicides.

If herbicide resistant oats are released commercially, it seems inevitable that hybridisation between wild oats and herbicide resistant varieties will occur. Simulation models (Barr, unpublished), similar to those described by Maxwell et. al. [12] have been used to predict the frequency of herbicide resistant, wild type individuals in crop systems where resistant oats and susceptible wild oats co-habitate. Estimates of wild oat fecundity in sprayed and unsprayed fields[13,14], an outcrossing frequency of 0.5% and knowledge of the genetics of weediness (Table 2) were included in the model. The rate of increase in frequency of resistant types can be reduced by 10 to 50 fold provided the recommendations in Table 3 (including selecting cleistogamous or low "promiscuity" varieties and always applying the AOPP herbicide to reduce wild oat populations in-crop) are implemented (Barr, unpublished).

Morrison and co-workers in Manitoba have judged the risk to be too great and Brown et al., (pers. comm.) despite having developed advanced germplasms[6] have abandoned the herbicide resistance program.

The rate of gene introgression into wild relatives could alternatively be measured in other systems where the carrier survives and non-carriers do not. One such example is the Grey Winter gene for oat stem nematode (Ditylenchus dipsaci) resistance. A resistant, cultivated type and susceptible wild oat (treated with nematicide) could be sown into a nematode infested field and the frequency of resistant, wild types monitored in subsequent generations.

Management of Herbicide Resistant Varieties

There are some areas and farming systems of oat production where wild oats are not a major weed and hence herbicide resistant varieties should be developed as soon as practical to facilitate control of other grassy weeds. For example, risks associated with oat ensilage and hay and especially forage, are very low because either opportunities for hybridisation or seed production are limited.

Management of agricultural systems infested with wild oats are more difficult especially with oat grain crops since the chances for hybrids to occur and of their progeny to survive are high. Judicious management plans will be required to reduce the build up of resistant weedy oats and maximise the value of any breeding program for resistance (Table 3).

Ideally, resistance to a herbicide from a group which is declining in importance in the major crops should be transferred to oats. Then, if resistance introgressed into wild oats, the options for control would not be seriously diminished. However, most of the current research and resistance genes are to herbicides inhibiting acetyl CoA carboxylase. Only Kibite and Harker (this volume) have characterised high level resistance to other groups.

Table 3. Management strategies to reduce the likelihood of wild oat x cultivated hybrids and the survival of their progeny.

Year	Management Target	Strategy
1. Preceding crop	All rotational crops	1. Reduce density of A. sterilis and A. fatua
2. Herbicide resistant oat crop	Oat crop	1. Breed "low risk" variety 2. Use AOPP herbicide to reduce wild oats 3. Do not save seed from fields heavily infested with wild oats 4. Amended statutory seed certification rules to reduce limits on wild oat admixture
3. First crop after oats	F1	1. Kill early germinations by cultivation or non selective herbicides prior to sowing 2. Apply CHD, thiocarbamate or other selective graminides in-crop 3. Do not use AOPP herbicides
	Rotation crop type	1. Choose highly competitive crop such as Canola where a wide range of graminicides can be used; do not grow oats
4. Second crop after oats	F2 plants	1. Do not use AOPP herbicides 2. Use a different, non-AOPP, graminicide to that chosen last year 3. Encourage germination; kill with non-selective herbicides
	Rotation crop type	1. Choose a different crop where different graminicides can be used. 2. Choose a crop where cultural control of wild oats is possible e.g. Sorghum, hay, pasture, late sown safflower/sunflower. 3. Do not grow oats

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The Conservation and Exploitation of Wild Oat Species

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Summary

Primary and secondary habitats of the wild *Avena* species are being eroded by man's increasing demands on the land for food production, which will ultimately lead to the irreplaceable loss of genetic variation of the genus *Avena.* The ease of exploitation of this genetic variation is dependent upon which of three gene pools the donor species belongs to.

Conservation

Worldwide collections of all our major crop plants have been made during the last two or three decades and many thousands of accessions of wheat, barley, maize and rice etc., have been amassed. Collections of wild *Avena* have also been made, but are very small in comparison. Having identified this discrepancy, the International Board for Plant Genetic Resources has sponsored a number of collecting expeditions. This followed the recommendations of the European Cooperative Programme for the Conservation and Exchange of Plant Genetic Resources to collect particular species which were poorly represented in existing world collections, and to attempt to assess any threat of genetic erosion, either at present or in the future.

In the last decade, a new diploid taxon *A.atlantica*[2] and a new tetraploid species *A.agadiriana*[3] have been collected in Morocco. Similarly, accessions of the diploid species *A.prostrata*, and *A.damascena* previously known only from Spain and a single site in Syria respectively, were collected in Morocco during 1985 and 1988[6]. From the diversity of species and taxa collected in North Africa and other countries bordering the Mediterranean, it is evident that this region can be regarded as an area of great importance as a source of genetic variation for the oat crop and it seems likely that there are further species/taxa of *Avena* to be found in this region.

Observations made during these collecting expeditions have indicated that many of the primary and secondary habitats of these wild weedy species are being eroded. Changes in farming practice and systems is a major factor contributing to the erosion of wild *Avena* as developing nations intensify their agriculture and the developed nations find alternative uses for the land. For example, the area in Spain where Ladizinsky collected in the early seventies[5], has now been taken over by the glasshouse industry to grow vegetables for the European market and has led to the loss of large areas of primary habitat. The remaining areas of heavy alluvial soil which at present are mainly permanent pastures supporting a rich and diverse flora including populations of the tetraploid oat *A.murphyi*, will undoubtedly be ploughed to give way to more lucrative cash crops.

In Morocco, which can be considered an area of diversity of the genus[6] the increasing human population is making heavier demands on the land, the consequences of this being greatly increased grazing pressures, which will rapidly lead to the annihilation of annual plant species, not least the Avenae. Further to this, if the predictions with regard to global warming are correct, leading to an even more rapid northward movement of the deserts, the loss of primary and secondary habitats will be greatly accelerated.

Genetic Variation

As can be seen from the limited examples listed in Table 1, tremendous genetic variation exists within the wild species of *Avena* for resistance to pests and diseases, as well as other characters which would greatly improve the cultivated oat crop if gene transfers could be effected.

Table 1.   Some of the useful variation identified in wild *Avena* species.

| | Resistance to: | | | | | Genes for high: | | |
	MIL	CR	SR	BYDV	NEM	OIL	PROT	CT
Diploids:								
A.canariensis					+		+	
A.damascena	+							
A.eriantha	+							
A.hirtula	+							
A.prostrata	+							
A.strigosa	+	+	+	+	+		+	
A.wiestii					+			
Tetraploids:								
A.barbata	+	+	+	+	+			
A.ludoviciana			+					
A.macrostachya				+				+
A.maroccana		+		+	+		+	
A.murphyi	+				+		+	
Hexaploids:								
A.sterilis	+	+	+	+	+	+	+	+
A.fatua				+			+	+
A.occidentalis				+				

Key: MIL = Mildew;   CR = Crown rust;   SR = Stem rust;
     BYDV = Barley yellow dwarf virus;   NEM = Cereal cyst nematode;
     OIL = Oil content;   PROT = Protein;   CT = Cold tolerance.

The sample data presented in Table 1, do not truly reflect the variation available in the wild weedy species of *Avena*. As will be seen below, gene transfers from *A.sterilis* to the cultivated oat are relatively straightforward, and consequently accessions of this species have been widely screened for desirable genes. There is little doubt however, that a similar diversity of variation exists in all the *Avena* species.

Gene Pools and Gene Transfer

The wild species can be grouped into three categories or gene pools[4] depending on the ease of gene transfer to the cultivated crop:

Primary

In this gene pool there is a free flow of genes between the wild and cultivated species, and the selected trait can be recovered by a conventional backcrossing programme. All the wild hexaploid taxa fall in this category e.g. *A.sterilis*, *A.fatua* and *A.occidentalis*.

Secondary

Here, gene flow is partly restricted, in that F1 hybrids can be readily produced, but are self sterile. Backcrossing these F1 hybrids to the recurrent parent however does produce a small number of seeds since they are partially female fertile. As the number of backcrosses increases, so the fertility levels increase. The tetraploids *A.murphyi* and *A.maroccana* are the only taxa within this gene pool.

Tertiary

Gene flow in the tertiary group is very restricted. This category includes all the diploid species and the tetraploids other than *A.maroccana* and *A.murphyi*.

Gene transfers from the tertiary gene pool to the cultivated oat are more difficult. Producing the F1 hybrid normally requires mass pollination followed by embryo rescue. Resultant F1 hybrids however are completely sterile. Doubling the chromosome number by treatment with colchicine invariably restores some degree of fertility, thus allowing backcross generations to be produced. In this way, addition lines with forty two chromosomes from *A.sativa* and two from the alien species, can be assembled and selection for the line containing the required gene(s) implemented. Experience has shown however that chromosome addition lines have a tendency to break down due to meiotic instability[8]. Occasionally, there is sufficient homology between the chromosomes of the cultivated oat and the added wild chromosomes to permit limited recombination, but such events are infrequent due to the large structural differences between chromosomes.

Where natural recombination fails, ionizing radiation or chemical mutagenesis can be used to effect recombination[1] but lines derived from such treatments, invariably suffer from duplications and or deletions of vital chromosome segments. Homoeologous chromosome pairing can be induced using the genotype CW57 of *A.longiglumis* which suppresses the mechanism preventing the pairing of homoeologous chromosomes[9]. This system has been used to good effect in transferring genes for mildew resistance from the tetraploid *A.barbata*[13].

Chromosome substitution lines can provide a better alternative to addition lines. They are produced by crossing the requisite disomic addition line with the monosomic series and selecting the line in which the substituted alien chromosome compensates (usually very specific[10,11]) for the lost *A.sativa* chromosome pair. Because of the problems associated with alien chromosome addition and substitution lines, no oat variety have been produced using these techniques, but they can serve as starting blocks for other cytological procedures.

Conclusions

Whilst it is true to say that the majority of agronomically desirable characters transferred from the wild to the cultivated oat have been derived from the hexaploids *A.fatua* or *A.sterilis*, this variation is not limitless. The time will come when we will have to divert greater attention to the tetraploids and diploids which possess a wealth of genetic variation waiting to be exploited, even though the transfer of such variation can involve long and protracted cytological procedures. We must ensure that when that time comes, we are not only able to exploit them, but that we have them conserved for exploitation.

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Oat Improvement with Genes from *Avena* Species

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Summary

The germplasm used to initiate cultivated oats in Europe probably contained a relatively small sample of the genetic variability available in *Avena sterilis* L., the crop's most likely progenitor. In oats taken to the New World, this germplasm base was narrowed even further. Attempts over the past 50 years to expand the gene pool of cultivated oats by transferring genes from wild oat species are reviewed in terms of disease resistance, grain quality, plant vigour and grain yield.

Introduction

The domestication of oats in Europe probably occurred less than four millenia ago[8] from *A. sterilis*[7,9] carried to Europe from the Middle East as a weed in cultivated barley and wheat. Most likely, the selection of a few nonshattering plants of *A. sterilis* from wheat and barley fields initiated oats as a cultivated crop. If this were the case it would have resulted in a narrow germplasm base for this crop in Europe.

As migration took place from Europe to new continents in the 1600-1800's, oats and other crops were carried by the settlers to these new lands. According to Coffman[9], most of the oat germplasm utilized and developed in the USA until 1970 traced to seven land cultivars introduced from Europe. Red Rustproof and Winter Turf have been the sources of red oat cultivars used in the southern USA, and most spring oat cultivars trace to Kherson, Green Russian, Victory, Markton, and White Russian. Pedigrees of American, Chilean, and Canadian oat varieties given by Forsberg and Shands[14] show that these land race introductions provided the genetic base for cutivars developed throughout the New World.

Obviously, the germplasm imported from Europe and utilized for oat breeding in the New World was very narrow. Although not documented, the same situation probably is true for oats on other continents where Europeans settled. Evidence for the narrowness of the cultivated oat gene pool is (a) the fact that all of its useful genes for crown rust resistance were consumed by 1960 and (b) the meager improvement in yield that occurred in the Corn Belt USA via breeding until 1975[5,25,32].

Over the past 50 years, many attempts have been made to transfer genes from the di- and tetraploid species to expand the gene pool of cultivated hexaploid oats[43,19,13,4]. Utilization of *A. fatua* and *A. sterilis* as germplasm donors to cultivated oats is routine because all hexaploid oat species share common chromosome morphology[29].

Disease Resistance

Suneson[39,40,41] developed Rapida, Sierra, and Montezuma oat cultivars from matings of *A. fatua x A. sativa.* Genes from *A. fatua* caused extreme earliness and large seeds. Also, Burrows (V. Burrows, pers. comm., 1992) developed "dormoats" by utilizing dormancy genes from *A. fatua.* Dormoats show "...improved grain yield, disease escape, early maturity, and improved grain quality...". Marhsall (H. Marshall, pers. comm., 1992) obtained winterhardiness from *A. fatua x A. sativa* matings that was equivalent to that of elite winter cultivars.

A. sterilis is the progenitor of cultivated oats and the USA World Oat Collection contains 5500 accessions of this species. Simons *et al.*[35] recognized that *A. sterilis* was an important source of genes for crown rust (*Puccinia coronata* f. sp. *avenae* Cda) resistance 30 years ago. Seven of 10 genes for crown rust resistance in Multiline E77[20] and eight of nine genes in Webster cultivar[22] were derived from *A. sterilis.* About 30 genes for crown rust resistance

from this species have been studied in Canada[36]. Crown rust resistance genes, Pc-38 and Pc-39 from *A. sterilis* have been used in the Canadian cultivars, Fidler and Dumont[28,27], and Pc-38 and Pc-39 and the gene for stem rust resistance, Pg-13, also derived from *A. sterilis*, have been used in the Canadian cultivars, Riel, Robert, and A.C. Marie (D. Harder, pers. comm., 1992). Crown rust resistance genes from *A. sterilis* have been used in the Texan cultivars, TAM 0-031, TAM 0-312, and TAM 0-396, and in eight cultivars developed by Coker's Seed Company, Huntsville, South Carolina USA. Don and Hazel cultivars from Illinois USA probably have crown rust resistance genes from *A. sterilis* (M. McDaniel, pers. comm., 1992). In contrast to the plethora of unique crown rust resistance genes found in *A. sterilis*, this species has contributed only two genes for resistance to stem rust (*Puccinia graminis* Pers. ex Pers. f. sp. *avenae* Ericks. & E. Henn.). A gene for resistance to mildew (*Erysiphe graminis* DC. f. sp. *avenae* Morch.), Eg-3, was found by Hayes and Jones[23] in an *A. sterilis* accession.

Grain Quality

Comeau[11] found tolerance to barley yellow drawf virus (BYD) in 9 of 20 *Avena* species, and of 1718 accessions of *A. sterilis* tested[10], 46% were tolerant to BYD. Comeau[10] states "....many of the recent oat cultivars in the USA and Canada derive some BYD resistance factors from *A. sterilis*....". McDaniel (M. McDaniel, pers. comm., 1992) says "It is likely that both McDaniel and Harrison (Coker Seed Co.) also have gained some BYD resistance from *A. sterilis* lines utilized as sources of crown and/or stem rust resistance."

Groat (caryopses)-protein contents from *A. sterilis* accessions have been reported from 27-35% [30,6,21]. Campbell and Frey[6] concluded that groat-protein percentage was simply inherited in *A. sativa* x *A. sterilis* matings, and Sraon et al.[37] estimated that from 1 to 25 factor pairs were segregating for groat-protein percentage in interspecific matings. Cox and Frey[12] reported that alleles for high groat-protein percentage from *A. sterilis* and *A. sativa* tend to be dissimilar and complementary. The mean heritability for groat-protein percentage in interspecific matings was 42% when the unit of selection was a spaced plant. Kuenzel and Frey[24] found five matings that involved genes for high groat-protein percentage from the two *Avena* species in which the genetic correlation between grain yield and groat-protein content was zero. McFerson and Frey[26] used five lines from these matings to initiate a recurrent selection program to increase groat-protein yield. Over three cycles of recurrent selection, the gain in protein yield was 27 kg ha^{-1} cycle^{-1}. Groat yield and groat-protein content increased 95 kg ha^{-1} cycle^{-1} and 0.31% per cycle, respectively, and the genetic correlation between these two traits was a non significant 0.13. This appears to be a unique source of germplasm in which these two traits can be increased simultaneously.

Groat-oil content in *A. sativa* was found to range from 4.2 to 11.3%[33] and in *A. sterilis* from 2.0 to 11.6%[21,31]. Groat-oil content is quantitatively inherited[42] and the genes for high-oil content from *A. sativa* and *A. sterilis* tend to be dissimilar and complementary[42]. In an oat population that contains high groat-oil genes from both species, Branson and Frey[2] found the mean groat-oil content to be 11.3% and the highest line had 14.5% oil after three cycles of recurrent selection. After six cycles, Schipper and Frey[34] found the population mean to be 14.2% oil and the highest line had 16.4% groat-oil. The ninth cycle is being evaluated in 1992. Third-cycle lines from this population were used to develop lines adapted in Scandinavia with 10% oil (B. Mattsson, pers. comm., 1992).

Grain Yield

In Iowa, extensive use has been made of genes from *A. sterilis* to increase vigor and productivity of cultivated oats. Frey and Browning[17] and Frey[15] showed that a crown rust resistance gene from *A. sterilis* accession CI 8079 caused an associated grain yield increase of 3.5 to 7.0 in disease-free environments. A number of lines developed from populations that contained from 3 to 13% *A. sterilis* germplasm had agronomic traits similar to the recurrent parents and grain yields 10-29% higher[16]. The superior yields of these lines were due to them having a leaf-area duration (LAD) greater than the recurrent parent[3,1]. The LAD superiority of 21-47% was caused by the derived lines having 10% larger leaf blades and delayed leaf senescence.

Several varieties with improved traits have been developed from matings involving *A. sterilis*. Hamilton cultivar, which was released in Iowa, is a BC$_2$F$_2$-derived line with *A. sterilis* cytoplasm. It yields 25% more than its recurrent parent and has better BYD tolerance. Sheldon cultivar, also released in Iowa, was an F3-derived line from a population with 12.5% *A. sterilis* germplasm, and Starter cultivar, released in Minnesota, has *A. sterilis* in its parentage.

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Genetic Resources of Tetraploid Wild Oats and their Utilization

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Introduction

The genus *Avena* contains four tetraploid species: the perennial, outcrosser and autotetraploid *A. macrostachya*, and the annual allopolyploids *A. barbata*- including the Ethiopian forms *A. abyssinica* and *A. vaviloviana*, *A. magna* and *A. murphyi*. Domesticated oats include diploid and hexaploid types but no tetraploids. The wild tetraploid species are isolated from the cultivated oats by crossability barriers in some cross combinations and high sterility of the interploid hybrids. As a result, the gene pools of the tetraploid and hexaploid species have evolved independently, causing some of the tetraploids genetic diversity to be absent in the hexaploid oats, including the domesticated types. The outstanding winter hardiness of *A. macrostachya*, the high protein content of *A. magna* and *A. murphyi*, and resistance of the tetraploid species to a large number of diseases are just a few examples. Despite the potential of the wild relatives as genetic resources plant breeders may utilise them in their programme only when the characteristic they are interested in is lacking in the cultivated gene pool.

A common procedure for utilising wild species, including the tetraploid oats, in breeding programmes is to backcross the interspecific hybrid to the cultivated plant in order to achieve chromosome stability and higher fertility, and to eliminate undesirable traits introduced by the wild species. A second approach for utilizing tetraploid species lies in the production of synthetic hexaploids followed by hybridisation to the cultivated oat. A third way, and probably the most radical one, is to domesticate the tetraploid species by transferring agronomic properties of the cultivated oat to them.

Factors Affecting Gene Transfer

Effective gene transfer between the cultivated hexaploid and wild tetraploids depends upon a number of characteristics which are essential in any of the available methods.

Crossability

The four tetraploid oat species are usually crosscompatible with the cultivated hexaploid oat when the latter is used as the female parent. Sadanaga and Simons[7] however, reported that in a cross with *A. abyssinica*, hybrid seeds were obtained when the tetraploid parent served as the female. While cross direction poses no restriction on the transfer of nuclear genes, it may prevent utilization of the tetraploid species cytoplasmic diversity by conventional crossing techniques. In crosses involving *A. barbara*, *A. magna* and *A. murphyi* the hybrid seeds are usually smaller than normal, but they germinate with no difficulty. Hybrid embryos involving *A. macrostachya* abort about two weeks after fertilisation but can be rescued via embryo culture[6].

Genetic recombination

Although hybrids involving the four tetraploid wild oat species and the cultivated hexaploid oat are all vegetatively normal, the pattern of chromosome association at meiosis and the number of chiasmata in these hybrids are key elements in the process of gene transfer. Mean number of chiasmata per cell is an indication of the genetic size and recombination potential of a species. This figure is also useful for comparing the potential of different species with the same chromosome number. For comparing recombination potential of species with different chromosome number, or interploid hybrids with their parents, mean chiasmata per cell can be transformed to association per chromosome (mean number of chiasmata per cell/n chromosome number).

The four tetraploids and the hexaploid oats have more or less the same number of associations per chromosome. The values of their pentaploid hybrids are much lower (Table 1) because a relatively large number of chromosomes are left unpaired, as univalents. In the pentaploid hybrid involving *A. macrostachya* some of the observed bivalents (theoretically seven) are a result of autosyndesisbecause the autotetraploid origin of this species. When these are not counted the mean chiasmata between chromosomes of the two parents becomes extremely low.

Table 1. Mean number chiasmata (Xta) in 4X and 6X *Avena* species and their hybrids

Species and hybrids	Univalents	Xta/cell	Xta/chromosome
Tetraploids (4X)			
A. magna		27.72	1.98
A. murphyi		26.88	1.92
A. barbara		26.48	1.89
A. macrostachya		26.11	1.87
Hexaploids (6X)			
A. sativa		41.58	1.98
Hybrids (5X)			
A.s X A.ma	8.10	21.00	1.20
A.s X A.mu	11.46	16.80	0.96
A.s X A.b	16.20	11.20	0.64
A.s X A.mac	15.43	15.99.	0.91.
		3.95[1]	0.22[1]

[1] excluding ring bivalents which probably resulted from autosyndesis of *A. macrostachya* chromosomes.

The degree of homology between chromosomes of the tetraploid species and *A. sativa* can be estimated by comparing the number of chiasmata (Xta) /chromosome and Xta/cell in the tetraploids species and in the pentaploid hybrids. Accordingly, *A. magna* has the highest and *A. macrostachya* the lowest homology with *A. sativa* chromosomes (Table 2).

Table 2. Proportions of the 6X and 4X genomes involved in 6X↔4X introgression.

Combination	Degree of homology[1]	% 6X genone affected[2]	% 4X genome affected[3]
A.s X A.ma	0.60	50	75
A.s X A.mu	0.50	40	62
A.S X A.b	0.33	26	42
A.s X A.mac	0.11	9	15

[1] $\dfrac{\text{Xta/ch 5X}}{\text{Xta/ch 4X}}$ [2] $\dfrac{\text{Xta/cell 5X}}{\text{Xta/cell 6X}}$ [3] $\dfrac{\text{Xta/cell 5X}}{\text{Xta/cell 4X}}$

Furthermore, the proportion of the hexaploid genome which may be affected by hybridisation with tetraploid species is indicated by the proportion of Xta/cell in the pentaploid hybrids as compared to the hexaploid parent. Here, again, introgression rate of a random gene to A. satira would be highest from *A. magna* and lowest from *A. macrostachya* (Table 2). In other words, introgression of economically valuable traits to the cultivated hexaploid oats by genetic recombination is most likely to succeed with *A. magna*. However, since the location of useful genes on the chromosomes of the tetraploid species is unknown it would be reasonable to try gene transfer from other tetraploid species whenever they possess such genes.

Sterility

Introgression from the wild tetraploids to the cultivated hexaploid oat is restricted by severe sterility of the pentaploid hybrids, resulting from irregular chromosome pairing at meiosis. In fact these hybrids are practically male sterile because their anthers do not dehisce at anthesis. However, a few seeds may be set by backcrossing them as female parent to either parent. The resultant seeds constitute a small fraction (less than 1%) of the florets produced on these hybrids making hand-crossing ineffective for obtaining a large number of backcross seeds. An alternative and labor-saving approach to obtaining these seeds is to grow the pentaploid hybrids among plants of the pollen donor parent and to allow natural cross-pollination to occur.

Stabilization of chromosome number and fertility

Viable gametes on the pentaploid hybrids exhibit a wide range of chromosome numbers, n=12 to 30 and n=14 to 27, in *A. sativa X A. murphyi* and *A. sativa X A.magna* respectively[5]. This indicates that gamete viability depends upon specific chromosome combinations rather than their total number in the gamete. Fertility was considerably improved in the backcrossed plants but not always correlated with euploid chromosome numbers. Stability of chromosome numbers and fertility was noticed in F_2 and at that stage plants with acceptable agronomic traits could already be selected[2,11].

The Backcross Method

Using this method, Sharma and Forsberg[8], attempted to transfer crown rust resistance from *A. abyssinica* to an *A. sativa cultivar.* Even in 2n=42 derivatives the resistance segregated because of the *A. abyssinica* substitution chromosome which was left unpaired. With the aid of thermal neutron irradiation Sharma and Forsberg[9] induced translocation of the *A. abyssinica* chromosome segment carrying the crown rust resistant gene into the *A. sativa* chromosome.

Thomas *et al.* [11] and Hagberg[2] utilized the backcross procedure to exploit the genetic diversity of *A. magna.* In $BC_1 F_3$ and $BC_2 F_2$ Thomas *et al.* [11] detected *A. sativa-* like segregants with protein content close to that of the *A. magna* parent. Only a moderate increase in protein content was detected by Hagberg[2] among $BC_1 F_8$ lines but they exceeded the cultivated parent in total grain weight and)ower hull percentage. In addition, extremely early-ripening was introduced from *A. magna.*

Synthetic Allopolyploid Bridge

An alternative approach to transferring genes from the wild tetraploid species to the cultivated hexaploid oat is to cross the tetraploids with *A. sativa*, to double the chromosome number of the pentaploid hybrids and to cross the synthetic polyploid with the cultivated oat. Using this method, Thomas *et al.* [10] attempted transferring a gene for mildew resistance from *A. barbara*. In F_4 they selected a 2n=44 resistant plant which was a disomic addition. To incorporate this gene into *A. sativa* chromosome irradiation was necessary[1].

Kummer[3] utilized *A. magna* to produce allohexaploids with the diploids *A. longiglumis, A. strigosa,* and *A. brevis,* prior to the crossing with *A. sativa,* and obtained transgressive

segregation for plant height and maturity and segregants with higher grain protein precentage than the recipient cultivated oat variety.

Domesticating the Tetraploid Species

Since gene transfer between the cultivated hexaploid oat and the tetraploids *A. magna* and *A. murphyi* is possible in both directions[5], the genetic diversity of these tetraploids can be exploited by transferring a considerable portion of the cultivated oat's agronomic properties to them. Theoretically, about 75% and 62% of the *A. magna* and *A. murphyi* genomes, respectively, can be affected by such a massive gene transfer (Table 2). While the need for domesticated tetraploid oats is not obvious at the moment, their impact on oat production and breeding could be as follows:

1. After appropriate breeding work, tetraploid oats could become a crop in their own right. This is analogous to wheat, where the tetraploid *durum* type is grown over a vast area, and for specific purposes. Wild *A. magna* and *A. murphyi* are exceptionally rich in protein and it is likely that their domesticated derivatives would have higher protein content than the hexaploid varieties. In addition, in certain areas and ecosystems tetraploid oats may grow more successfully than the hexaploid types.

2. Domesticated tetraploid oats could be the cornerstone in the production of synthetic hexaploid oats with novel genomic combinations. The most promising synthetic hexaploid might involve the cultivated diploid *A. strigosa*, because it possesses many agronomic attributes and its chromosomes exhibit preferential pairing in the presence of alien chromosomes[4]. Such pairing would ensure regular meiosis in the synthetic hexaploid and high fertility.

3. The domesticated tetraploids might also be used as a bridge for gene transfer from diploid species to the cultivated hexaploid oat. Hybridization between diploid and hexaploid oat species is difficult and usually requires embryo culture. However, these diploids are usually readily crossed with the tetraploid *A. magna* and *A. murphyi* as are the synthetic hexaploids with *A. sativa*. In addition, the common genetic background of the domesticated tetraploids and cultivated hexaploid varieties would facilitate selection of desirable recombinants.

We have recently renewed our effort to produce domesticated tetraploids and some of the results are reported below.

Production of domesticated tetraploids

To produce these tetraploids we followed the backcross method using the tetraploids as recurrent parents. In selecting parents for the initial crossing programme we took into consideration the very high sensitivity of both *A. magna* and *A. murphyi* to the viral disease BYDV and selected *A. sativa* lines with outstanding tolerance to this disease. In this hybridisation experiment it was also essential to eliminate, as quickly as possible, the spikelet-shedding base of the wild tetraploids. Spikelets of the wild tetraploids are shed upon reaching maturity leaving a large disarticulating scar at the base of the dispersal unit, whereas spikelets of *A. sativa* remain attached to the pedicel. In hexaploid oats the disarticulation pattern is governed by a single gene with dominance of the cultivated type. F_1 hybrids of *A. sativa* and *A. magna* had spikelet disarticulation pattern similar to that of *A. sativa* and following backcrossing to the tetraploid parent about 50% of the progeny exhibited the wild type. Backcross plants with the cultivated phenotype spikelet base segregated in F_2 to wild and cultivated types in a 1:3 ratio, and by progeny test homozygous domesticated types were selected. The stable domesticated families were then crossed to the wild *A. magna* to test the chromosome arrangement of the domesticated tetraploids. Hybrid seeds were easily obtained, the plants exhibited normal meiosis and were fertile. Spikelet base was of the domesticated type and in F_2 segregation of 1 wild : 3 domesticates occurred. This segregation pattern indicates that also in *A. magna* a single gene controls spikelet disarticulation, as in *A. sativa*, and in both species it is located on homologous chromosome segments.

After a single hybridisation cycle with *A. magna*, three meiotically stable and fertile domesticated tetraploid families were selected. In addition to their non-shedding spikelets they had yellow glabrous lemmas with zero to one awn per spikelet, and as such were almost indistinguishable from *A. sativa*. Protein content in the groats of the Aa2-3 family was similar to that of the *A. magna* parent (Table 3), and transgressive segregation to kernel size was noted in Aa2-13 and Aa2-16 families. The three families were inferior to the cultivated parent in that they had higher proportion of hull in the seeds.

Members of these three families have been utilized as tetraploid parents in a second hybridisation cycle with *A. sativa*. The cultivated lines used in these crosses include dwarf types, naked oat, and others with outstanding tolerance to BYDV. The overall aim is to accumulate sufficient diversity in the domesticated *A. magna* to enable future breeding work within the domesticated tetraploids.

A single hybridization cycle so far has been completed with *A. murphyi*. Here, too, domesticated tetraploid derivatives have been selected which will be utilized as tetraploid parents in the next hybridization cycle.

Using domesticated tetraploids in hybridisation with *A. sativa* increases introgression effectiveness because all the derivatives are expected to share the domesticated spikelet-base type, and desirable segregants can therefore be selected from a larger number of plants.

Table 3.   1000 kernel weight, % hull in seed and % protein in the groat of *A. magna*, *A. sativa* cv. Ogle and their tetraploid derivatives.

Line	1000 kernel weight (g)	% husks	% protein
A. magna	19.5	53.9	24.8
Ogle	28.3	21.1	19.8
Aa2-3	21.6	36.3	24.5
Aa2-13	33.9	52.2	21.6
Aa2-16	39.9	40.1	19.4

Production of Synthetic Hexaploids

Hexaploid oats, wild and cultivated, are cytogenetically allopolyploids but their origin, i.e. the diploid and tetraploid species which participated in their formation are not yet known. It is also not clear whether the genomic combination of the hexaploid oats is the best for achieving optimal productivity and quality levels in the farmer's field. The tetraploid domesticated types which have already been selected, and others which will be produced in the future, will enable the production and testing of new synthetic hexaploids which combine these tetraploids with different diploid species.

Domesticated derivatives of *A. magna* were crossed with the diploid oats *A. strigosa* cv. Saia and *A. longiglumis*. Chromosome number of the triploid hybrids was doubled by placing vials containing 0.02% colchicine solution on cut tillers of the young plants. The treated plants produced a few seeds, all with 2n=42.

(*A. strigosa* cv. Saia X Aa2-3)^2

Plants of this synthetic hexaploid were vegetatively normal, produced many tillers and panicles with 53 to 103 spikelets per panicle, and usually two florets per spikelet. All the spikelets had the domesticated base. Chromosome association at metaphase I was mainly in bivalents, and rarely 1 to 2 quadrivalents per cell were observed. Pollen fertility was usually above 85%, with seed set of 0.88 to 0.95 seeds per floret. One thousand seed weight was in the range of 44 to 53g, with the hall making up about 35% of this.

$(A.\ longiglumis\ X\ Aa2-16)^2$

These synthetic hexaploids were also vegetatively normal but taller than plants of the previous combination. However, they had less spikelets per panicle (50 to 75) and 2 to 3 florets per spikelet. Here, too, the spikelets had the domesticated-base type. Chromosome association at meiosis was less regular and cells with 1 to 3 quadrivalents were common. Pollen fertility ranged from 65 to 85% and seed set from 0.38 to 0.82 seeds per floret. Seeds were exceptionally long and weight of 1000 seeds ranged from 58 to 75g, 55 to 65% of this being hull.

The performance of the synthetic hexaploids indicates that in the future it should be possible to produce, in a single step, stable hexaploid varieties which combine the agronomie properties of the domesticated tetraploids and desirable traits of diploid species.

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Wild Oat as a Source of Disease Resistance: History, Utilization, and Prospects

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Summary

The earliest documentation of variation in disease (smut and rust) reactions by oat varieties or species occurred in the late nineteenth and early twentieth centuries. Resistance was found mainly in lower ploidy species, thus was not utilised in breeding. In the mid 1950's the value of resistance in natural populations, including hexaploids, was demonstrated. Beginning in 1964, a series of plant collecting expeditions to mid-eastern and Mediterranean regions have yielded numerous accessions with resistance to a range of oat diseases. Genes for resistance to crown rust, stem rust, smut, powdery mildew, and tolerance to barley yellow dwarf virus from wild hexaploid accessions have been isolated and utilised. A large reservoir of resistance to these diseases occurs in the lower ploidy accessions. Resistance has been found in accessions obtained from a broad geographical range. There was some regional correlation with resistance to a particular disease or ploidy level. Important to providing durable resistance is to have sources with a large diversity of resistance, either as multiples of unique seedling/adult genes or genes governing rate-limiting forms of resistance. The full range of resistance in all of the *Avena* accessions is not yet known, but the data so far indicates a great deal of useful diversity. Although the wild *Avena* spp. contain a rich source of resistance, there are some concerns regarding the future potential. The virulence plasticity of the pathogens is an important factor, since changes in crown rust virulence have already reduced the effectiveness of a number of genes. New resistance may be acquired through more collecting expeditions, but more returns would likely be gained by increasing and concentrating resources to develop sources in existing collections, either through use of current technologies or developing new and more efficient technologies to transfer resistance into agronomically viable oat types.

Introduction

Although considerable resistance from cultivated oat germplasm has been exploited to control oat diseases, its effectiveness has often been limited due to changes in pathogen virulence, or resistance to some important diseases was unavailable. Workers have long recognized wild or weedy *Avena* spp. as sources to supplement the resistance base, and indeed these sources now are an important component of present-day breeding efforts[30]. The continued value of wild *Avena* collections, however, will hinge on developments in pathogen virulence and in the knowledge and resources that will be available to access known or new sources of resistance. In this paper we will attempt to provide an overview of the history of wild oat as related to disease resistance, ascertain the extent of resistance from published and unpublished sources, its accessability, and evaluate its future potential.

Historical

Records of variation in infection of oat varieties or species began about 1888 with the account by Jensen[43] of resistance to loose smut in *A. strigosa*. Carleton[12] appears to have first documented rust resistance in *A. sterilis*. Subsequently other early workers in North America[59,60,61,62,83], Europe[26], and Russia[42,87] reported variations in resistance in several *Avena* spp. Much of this early work was aimed at assessing physiologic variation in the smut and rust pathogens, but Vavilov[87] in particular stressed the importance of these sources in resistance breeding. Most of the resistance was found in the lower ploidy accessions, thus

utilization was limited. The subject remained relatively dormant for some time, but an important step was made by Vavilov[88] with his theory that plant populations would show the greatest variability with respect to factors such as disease resistance at their centres of origin. Wahl in 1958[89], noting high rust pathogen virulence in Israel, emphasized the significance of the co-evolution of indigenous host/pathogen systems, resulting in enhanced host "tolerance". Then Dinoor and Wahl[24] suggested that these indigenous populations, including hexaploids, may have high levels of resistance to non-indigenous pathogen populations, a concept further elaborated by Dinoor and Eshed[23]. Heath[40] also theorized that host populations under long-term pathogen pressure evolve a broad base of resistance. Various studies[22,24,56,78,81] have confirmed the value of the Israeli collections for disease resistance in other parts of the world. Despite the positive results from the Israeli collections, the base of resistance germplasm, particularly against the rusts, still remained inadequate. The optimism, however, was sufficient to spur additional collecting expeditions to the Mid-East and to other regions encompassing the Mediterranean basin.

Collecting expeditions were initiated by the Canada-Wales project beginning in 1964 and ending in 1970, covering the Mediterranean region and many countries of the Middle-East[7]. Dr. J.W. Martens, Agriculture Canada, Winnipeg, subsequently made collecting trips to East- and North-African countries and the Canary Islands in 1972, and to Spain, Portugal, Morocco, and Canary Islands in 1981. Dr. A. Comeau, Agriculture Canada, Ste-Foy made additional collections from Turkey in 1981 with specific interest in barley yellow dwarf virus resistance[20]. The Canadian Avena (CAV) collection now numbers about 8500 accessions, of which over 5000 have been screened for rust resistance at the Winnipeg Research Station (Table 3). Other more recent collecting expeditions include those of Forsberg and Simons[28] from the USA into Turkey in 1986, and Hagberg[33], Leggett, and Ladizinsky to the western Mediterranean region in 1985. Frey[30] indicates that about 5500 accessions of A. sterilis alone are held in the USA (a number of these overlap with the Canadian Avena collection). Smaller collections of Avena spp. are held in many institutions around the world.

Species Relationships within *Avena*

To understand the extent and usefulness of resistance in Avena germplasm, it is important to understand the species/reproductive relationships within the genus. Various treatments[4,27,48,58] of oat taxonomy have included morphological, geographical, or reproductive relationships to arrive at species differentiation. Species have been determined on morphological features governed by as little as single gene differences[17]. To assess the distribu-tion of disease resistance within Avena, distinctions based on features other than reproductive compatibility or perhaps geographic isolation, are meaningless. A summary of oat species and their genomic relationships is given in Table 1 (derived from various studies)[5,6,50,51,58,85]. Species within genomic groups are generally interfertile, although minor modifications within the A and C genomes may result in cross-incompatibilities at the diploid level. Within Avena, reproductive barriers are normally due either to cross-incompatibility or sterility in the F_1[58].

Distribution of Resistance

An overview of selected reports of resistance to oat diseases or pests in non-cultivated oat species is given in Table 2. Some species like A. abyssinica, A. byzantina, and A. strigosa, although cultivated, also tend to be weedy and develop wild populations, thus were included. The most important diseases of oat on a world-wide basis are the rusts, smuts, powdery mildew, and barley yellow dwarf (BYD)[36]. Resistance or tolerance to these diseases has readily been found in a range of Avena species.

The rusts

The destructiveness of the rusts and reliance on resistance as the only practical means of control has been the driving force behind much of the North American Avena collection and research effort. The efforts have paid large dividends. So far 35 designated genes for crown rust resistance have been isolated. In addition, complex additive resistance[37], and

"field" resistance[80] have been identified. Other non-designated genes likely occur in Iowa multi-lines[31,32] or in other cultivars[30]. A broad range of resistance occurs in the diploid and tetraploid populations (Table 3), which has not yet been exploited.

Table 1. Species of *Avena*, their ploidy level and genomic composition

Species	Genomic composition	Species	Genomic composition
1. Diploid, 2N = 14		**2. Tetraploid, 2N = 28**	
A. clauda	CpCp	A. abyssinica	AABB
A. eriantha		A. barbara	
		A. vaviloviana	
A. ventricosa	CvCv	A. agadiriana	
A. prostrata	ApAp	A. maroccana	AACC
		A. murphyi	
A. longiglumis	AlAl		
		A. macrostachya	CCCC
A. damascena	AdAd		
		3. Hexaploid, 2N = 42	
A. canariensis	AcAc	A. byzantina	AACCDD
		A. fatua	
A. atlantica	AsAs	A. sativa	
A. wiestii		A. sterilis	
A. hirtula			
A. strigosa			

Efforts at finding stem rust resistance have been less successful. To date only two seedling genes, Pg13[55] and Pg15[52] and one adult plant-effective gene, Pg17[35], have been isolated from A. sterilis. From lower ploidy oat, gene(s)Pg6/Pg7 have been designated in A. strigosa[25], and Pg16 in A. barbarta[10]. Gene Pg16[10] and sources from A. strigosa and A. longiglumis[66] have been transferred to the hexaploid level, but only Pg13 from A. sterilis is known to occur in commercial cultivars.

A large reservoir of untapped resistance to both stem and crown rust appears to remain in wild Avena accessions. An example is the screening work, largely unpublished, on the Avena accessions at Agriculture Canada in Winnipeg (Table 3), between 1965 and 1984. The identification of resistance was based on recognisable infection types, thus rate-limiting forms of resistance, which are known to also exist in the wild Avena populatons[80], are not included. Also for the purposes of Table 3, all species were grouped into their respective ploidy levels, space not permitting a more detailed species-based analysis. Similarly, the geographic breakdowns are broader than desired.

There was considerable variation in crown rust resistance, both on a regional and ploidy basis. The tetraploids (mainly A. barbata) generally represented a broad range of resistance in all regions except East Africa (where A. abyssinica and A. vaviloviana were dominant). The effectiveness of the A. barbata accessions is indicated by the proportion which had resistance to three or more isolates than those to one or two isolates only.

Table 2. Known sources of resistance or tolerance to oat diseases or pests in wild *Avena* species.

Disease or pest	Species	Selected References	Disease or pest	Species	Selected References
Crown or stem rust	2N - canariensis, clauda, eriantha, hirtula, longiglumis, prostrata, strigosa, wiestii	11,22,24, 33,52,66, 68,70,71, 72,73,74, 75,78,77, 78,79,81, 84,89,90, 94	Powdery mildew	2N - atlantica, eriantha, hirtula, longiglumis, prostrata, strigosa, venticosa	41,44,45, 46,71,76, 77
	4N - abyssinica, barbata, maroccana, vaviloviana			4N - agadiana, barbata, murphyi, barbata	
	6N - byzantina, fatua, ludoviciana, sterilis			6N - byzantina, fatua, occidentalis, sterilis	
			Septoria leaf blotch	6N - sterilis	75
Smut	2N - strigosa, wiestii	57,63,91	Pyrenophora leaf blotch	2N - strigosa	47,77
	4N - abyssinica, barbata			6N - byzantina, sterilis	
	6N - byzantina, vaviloviana, fatua, sterilis		Barley yellow dwarf virus	2N - strigosa, 4N - barbata, macrostachya, 6N - fatua, occidentalis, sterilis	18,19,21, 64,82,94
			Oat mosaic virus	6N - sterilis	56
			Cereal cyst nematode	4N- maroccana	33

Table 3. Occurrence of resistance to *Puccinia coronata avenae* and *P. graminis avenae* in diploid, tetraploid, and hexaploid *Avena* species in five major geographic regions

Location[1]	P. coronata[2]								P. graminis[3]								
	1 isolate		2 isolates		3 or more isolates		Total No.	Total % ploidy	Total % Region	1 isolate		2 isolates		Adult Pl. res.		Total No.	Total % ploidy	Total % Region
	No.	%	No.	%	No.	%				No.	%	No.	%	No.	%			
1. Mid-East																		
diploid	5	85.0	3	5.1	0	0.0	59	13.6		1	1.1	1	1.1	1	1.1	89	3.4	
tetraploid	47	20.0	38	18.5	40	24.4	205	65.9		9	2.4	14	3.4	13	3.5	370	9.7	
hexaploid	218	12.9	82	4.8	56	3.3	1692	21.0	25.5	56	3.3	21	1.2	124	7.3	1694	11.9	11.0
2. N. Africa																		
diploid	11	15.1	16	21.9	30	41.1	73	78.1		4	3.4	9	7.8	6	5.2	116	16.4	
tetraploid	45	16.2	45	16.2	125	45.1	277	77.6		13	4.0	11	3.4	21	6.5	321	14.0	
hexaploid	86	8.5	48	4.8	46	4.6	4006	17.9	33.3	18	1.6	2	0.2	30	2.7	1124	4.4	7.3
3. Canary Is.																		
diploid	19	17.4	12	11.0	12	11.0	109	39.4		1	0.9	0	0.0	2	1.8	113	2.7	
tetraploid	28	15.1	28	15.1	100	54.0	185	84.3		7	3.5	1	0.5	2	1.0	200	5.0	
hexaploid	41	18.5	33	15.0	26	11.8	220	45.3	58.2	3	1.4	0	0.0	4	1.9	213	3.3	3.8
4. E. Africa																		
diploid	0	0.0	0	0.0	0	0.0	0	0.0		0	0.0	0	0.0	0	0.0	0	0.0	
tetraploid	1	0.3	2	0.7	2	0.7	287	1.7		7	2.3	22	7.4	78	26.1	298	35.9	
hexaploid	0	0.0	0	0.0	0	0.0	87	0.0	1.3	0	0.0	1	0.8	3	2.5	118	3.4	26.7
5. Iberia																		
diploid	14	21.0	6	9.0	47	70.1	68	98.5		3	3.7	7	8.6	3	3.7	81	16.0	
tetraploid	11	7.6	24	16.7	99	68.8	144	93.1		6	4.0	4	2.7	4	2.7	147	9.5	
hexaploid	55	19.5	54	19.1	121	42.9	282	81.6	87.2	2	0.6	5	1.6	8	2.6	312	4.8	7.8
Total No.					4694											5196		

[1] 1 = Iran, Iraq, Turkey, Israel, Syria, Lebanon; 2 = Algeria, Morocco, Tunisia; 4 = Kenya, Ethiopia; 5 = Spain, Portugal
[2] Resistance to either 1, 2, or 3 or more isolates
[3] Resistance to either 1 or 2 isolates, or adult plant stage only

Resistance in the diploids varied more by region, with relatively low incidence (13.6%) in the mid-East (represented mainly by *A. clauda*, *A. pilosa* (*eriantha*) and *A. wiestii*), moderate resistance (39.4%) in the Canary Islands (mainly *A. canariensis*), high levels (78.1%) in North Africa (highest in *A. longiglumis* followed by *A. hirtula* and *A. wiestii*), and very high levels (98.5%) in Spain and Portugal. Incidence of resistance in hexaploids (mainly *A. sterilis* at all locations) was low in the mid-East (21.0%), and North Africa (17.9%), moderate in the Canary Islands (45.3%) and high in Spain and Portugal (81.6%). Also, except for Spain and Portugal, the hexaploids tended to have a higher proportion of resistance to only one or two isolates than to three or more isolates.

Resistance to stem rust was much less common. Similar to crown rust, the poorest source in the mid-East was in the diploids, but in this region the hexaploids had higher incidence relative to the tetraploids. Similar ploidy/resistance relationships occurred in North Africa, Spain, and Portugal, but at much lower levels than for crown rust. There was very little resistance in the Canary Island accessions. The East African (mainly *A. vaviloviana* from Ethiopia) collection showed a higher incidence of resistance, predominantly at the adult plant stage. Adult plant resistance to stem rust was quite common throughout the accessions.

Loose and covered smut

In the early literature, Reed[59,60,61,62] and Williams and Verma[91] reported good sources of resistance in several *Avena* spp. Nielsen[57] tested 1674 accessions of six species of the Canadian *Avena* collection held at Winnipeg, and found 869 of these, mainly *A. abyssinica* from Ethiopia and *A. sterilis* from Ethiopia, Israel, Lebanon, Syria, and the North African countries, to be resistant. Resistance from an *A. sterilis* source first appeared in the cv. Fidler, released in 1981[54] and occurs in all oat cultivars released from the Winnipeg Research Station since that time. The resistance from *A. sterilis* continues to confer complete immunity to all known North American smut races (Dr. J. Nielsen, pers. commun.) and is easily transferred, thus provides an excellent breeding resource.

Powdery mildew

Resistance to powdery mildew in wild *Avena* species at all ploidy levels has been reported from Europe[41,45,70,71,76,77] and the USA[94]. Most of the resistance has been found in the lower ploidy accessions, and in one case was successfully transferred from *A. barbata* to the hexaploid level[1,2]. Transfer from the diploid *A. pilosa* (*eriantha*) has been less successful because of instability at the hexaploid level due to several *A. eriantha* chromosomes being involved in the control of resistance[71].

Barley yellow dwarf

Zillinsky and Murphy[94] first determined tolerance to the BYD virus in *A. barbata* and *A. sterilis*. Comeau[18] screened 1718 accessions of *A. sterilis* from 17 countries or areas, and was able to identify resistance or tolerance in 46% of these. Resistance was highest from Algeria, Tunisia, Lebanon, and Turkey, often associated with habitats favourable to the aphid vectors. Turkey was reinforced as a prime source of BYDV tolerance in further collections[20] from this region (A. Comeau, pers. commun.). Also, 9 of 20 *Avena* species showed tolerance, with the highest levels in *A. sterilis* and *A. occidentalis*, but high levels also occurring in *A. barbata*, *A. fatua*, *A. hybrida*, *A. machrostachya*, *A. nuda*, and *A. strigosa*[19]. Dr. S. Haber (pers. commun.) has found lines of *A. strigosa* virtually immune to BYDV. Resistance/tolerance to BYDV generally appears to involve 2 to 4 quantitative-effect genes[49,53]. Tolerance to BYDV has shown steady improvement in many North American oat cultivars in recent years[21], with much of this improvement likely a product of the extensive use of *A. sterilis* in rust resistance breeding.

Diversity of Resistance

Screening *Avena* populations for disease resistance may show high levels of resistance, but this may demonstrate only that one or a few genes, effective against the pathogen isolates used, are widespread within a reproductively compatible population.

To contribute to longer term stability of resistance, diverse sources of resistance are required. Diversity may refer to multiples of unique genes effective in adult or seedling plant stages, or to genes governing quantitative or rate-limiting forms of resistance. It is possible to assess diversity only if sufficient accessions are tested with a range of pathotypes, and to be certain, must be confirmed genetically. The diversity of resistance against the smuts and powdery mildew is difficult to assess. More meaningful assessments of diversity against the rusts, however, may be made due to the numbers of accessions tested (e.g. Table 3), using with a diverse range of pathotypes, and data on inheritance involving many of the isolated genes.

Although the incidence of resistance to stem rust was low in the CAV collection, there are indications of considerable diversity. Adult plant resistance appears to be common at all ploidy levels (Table 3), and an adult resistance gene (*Pg17*) from *A. sterilis* has been documented[35]. Various levels of rate-limiting or quantitative resistance to stem rust in the hexaploid CAV accessions is indicated, but remains to be verified. In a genetic study involving 10 stem rust resistant tetraploids, including the source of *Pg16*[3], 8 accessions segregated when crossed with a *Pg16* line, and one did not. The eight segregating lines also appeared to be diverse resistance genotypes, as judged by reactions to a range of rust fungal isolates[3].

In their tests for crown rust resistance in wild *Avena* populations, Dinoor[22], Dinoor and Wahl[24], and Simons[78] indicated the potential resistance diversity. Subsequent work has indeed indicated an enormous range of crown rust resistance diversity in *Avena*. Where genetic studies have been undertaken, in only a few cases have gene duplications been indicated (*Pc36/47*, *Pc40/49*[79], a gene in *A. sterilis* from Spain = *Pc68*[35], and a gene in CAV 3695 = *Pc50*[38]). Fox[29] has isolated 6 genes from *A. sterilis*, which on preliminary analysis appear to be unique. The genes for resistance isolated so far from *A. sterilis* are random selections representing the "best" potential resistance from screening tests, but many other promising selections exist. Thus the full range of simply inherited crown rust resistance genes occurring in this species is still not known. In addition, adult plant resistance[39], complex additive resistance[37] and genes for 'field' resistance[80] have been characterized. Further indication of diversity in the CAV accessions is shown in Tables 3 and 4. In Table 3 the separation of isolates into categories with resistance to 1, 2, or 3 or more isolates is a preliminary indicator of diversity. The reactions of 144 tetraploid and 282 hexaploid accessions, all from Spain, to 6 isolates of *P. coronata* are shown in Table 4. The tetraploids differentiated into 20 unique infection patterns of resistance/susceptibility, and the hexaploids into 24 patterns. In some cases resistant-only patterns were further differentiated by unique infection types. These results indicate a great deal of diversity for resistance within a relatively small geographic region.

Future Potential

The work done to date world-wide has shown that non-cultivated populations of *Avena* are rich sources of disease resistance, much of it still untapped. What is the future? The first question is, do our present collections adequately represent the resistance available in *Avena*? This is difficult to answer since the screening and genetic analysis of existing collections is far from complete. We have sufficient information regarding geographic distribution of resistance to the most important diseases, and given the apparent diversity, more intensive collections would likely yield new resistance, although probably with diminishing returns.

A second important question is the relation of resistance to pathogen virulence dynamics. For diseases such as BYD and the smuts, rapid changes in virulence are not known to occur. The stem rust pathogen may undergo rapid changes, but in recent decades has been very stable in North America[34,65]. With crown rust, the situation is more disquieting. This pathogen is enormously plastic in its virulence, with rapid evolution of new virulence combinations. The *Pc38/Pc39* gene combination has been the cornerstone of resistance in the northern plains of North America, and no evidence of virulence to this combination appeared world-wide for many years. However, virulence appeared in Australia in 1979/80[9], then in Canada[13,14] and now comprises a substantial proportion of the crown rust population in the northern plains of North America, in a number of different virulence combinations[15].

Table 4. Diversity of resistance to *Puccinia coronata avenae* in accessions of tetraploid and hexaploid *Avena* species collected from Spain, as indicated by range of infection types to 6 isolates of *P. coronata*

Isolate[1] of *P. coronata* and infection type											
Tetraploid *Avena* (144 accessions)						Hexaploid *Avena* (282 accessions)					
CR32	CR36	CR107	CR50	CR25	CR77	CR32	CR36	CR107	CR50	CR25	CR77
;0	;	;	0;	;1	;1⁻	4	4	4	;	4	3⁺4
2	2	2⁺	2⁻	;1	;1⁻	;	0;	2⁺	;	4	4
3⁺	4	3	;1	;1⁻	4	;	;	;	;1	4	33⁺
0;	0;	;	3⁺	3⁺4	3	;1	;1	;	;	;1⁺	1
;	4	;1	3⁺	4	1⁻	;	4	;1	3⁺	4	1⁻
2⁺	2⁺	3	3	3⁺	3	0;	;	;	0;	0;	0;
2	0;	0;	0;	;	;1	4	0;	4	4	4	3⁺4
3⁺	3⁺	;1⁻	;1⁻	;1	3⁺	4	0;	4	0;	4	4
2⁺	;1	3	3	;	;1	3⁺4	4	4	4	4	4
;1	;1	2⁺	0;	;1⁻	;	4	4	3	3⁺	4	2⁺3⁻
2	3⁺	2	2	4	4	4	0;	;	3⁺	3	4
4	0;	4	0;	4	4	4	3	;	;1	4	4
;1	;1⁻	;	;	4	1⁺	4	0;	4	4	4	;1⁻
;	;	3⁺	3	4	4	3	2⁺	3	3	4	2
3⁺4	3⁺	4	0;	4	4	;	;	;	0;	;1⁺	2⁺3
3	3	;1⁻	;1	2⁺	2⁺	;1	4	4	4	4	3
;1	1	;1⁻	;1	1⁺	4	;2	;	;1⁻	;1	4	2⁺3
;1	;	;1⁻	;1	4	3⁺4	;	;	;	;	4	;
4	0;	;1	;1	0;	12	;1	;1	;	;1	;	4
23	3⁺	;1	;1	;	;2⁻	3⁺	4	;2	;1	;	;
						3⁺	;1	3⁺	3⁺	4	2
						0;	0;	4	0;	4	;1⁻
						;	;	;	;	4	3⁺4
						3	1	3	1	3⁺4	4

[1] The isolates are CR accession numbers held at the Winnipeg Research Station, selected for their range of virulence to the known genes for resistance to *P. coronata*

Similarly, virulence to gene *Pc68*[93], which had conferred immunity to all known North American crown rust isolates, has already been found[16]. It is significant that gene *Pc68* has not yet been exposed to any portion of the North American *P. coronata* population. Earlier optimism of an extensive and effective base of crown rust resistance in the wild hexaploids has become tempered with the knowledge that virulence to much of that resistance becomes evident quickly, and increases rapidly with the release of germplasm from these sources.

A third major point is the resistance available in the lower ploidy species. Although transfers to fertile hexaploids have been made[2,10,27,66,68,69,86], there still are many difficulties. Chromosome differentiation within and between *Avena* genomes has resulted in effective isolating mechanisms. Irregular meiosis readily results in alien chromosome substitution or addition lines, with the desired genes then located on the alien chromosomes. Problems continue due to lack of isolation of the desired gene segment, resulting in depressed agronomic performance[8] (also associated with gene *Pg16* - authors, unpubl.), or expression of resistance[69] may be modified in the new background. Utilization of the lower ploidy germplasm will require the development of more effective technology.

A last but very important point regards the resources available for research. The past few decades have generally seen a decline, particularly in the public sector, in resources devoted to oat research. New germplasm sources could be collected, but it is equally important that existing collections be more fully evaluated, catalogued, and maintained/rejuvenated. The work on germplasm collections requires intensive effort, but is falling short. More resources will be needed to accelerate the use of lower ploidy germplasm even with existing technology, let alone development of novel new technologies.

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Interactions Between Wild and Cultivated Oats in Australia

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Summary

In Australia, three species of wild oat form close interactive associations with *A. sativa*. These interactions may be either of a genetic or an epidemiological nature. Genetic interactions occur through the formation of fertile interspecific hybrids. Quantitative estimates of the frequency of outcrossing between *A. fatua* and *A. sativa* in four naturally occurring mixed populations varied from 0 to 0.93% (mean=0.65%). This has important long-term implications for the development and release of herbicide tolerant varieties of cultivated oats. Epidemiological interactions occur because both cultivated and wild species are hosts to the same pathogenic organisms. Wild species may act as important pathogen refugia, carrying inoculum over from one crop season to the next. In addition, the existence of significant variation for resistance in wild oats to diseases like crown rust and stem rust, strongly suggests that the wild species place a diverse range of selective pressures on the pathogens favouring the emergence of a wide range of pathotypes. Pathogenic variability is greater in regions where variability for resistance in the wild oat populations is higher than in regions where resistance is low or absent.

Introduction

In Australia, three introduced species of wild oat (*Avena barbata, A. fatua* and *A. ludoviciana*) have the potential to form close associations with their crop relative. As contaminants of cereal crops and as weeds of rural roadsides, wild oats often grow in close juxtaposition to cultivated oats (*A. sativa*). In this situation there is the potential for genetic interaction between some of these species. However, close spatial proximity is not an essential ingredient to the occurrence of interactions between wild and cultivated oats. All three wild species are hosts to the same pathogens that attack cultivated oats (for example, *Puccinia coronata, P. graminis avenae*, barley yellow dwarf virus) and as components of roadside vegetation and pastures subject to low grazing pressures, may form extensive stands covering hundreds of hectares. The existence of these stands has significant implications for the epidemiology and microevolution of such pathogens.

Wild Oat — Cultivated Oat Interactions

Genetic exchange between species

Cultivated oats and the three species of wild oat occurring in Australia can be classified into two chromosome groupings. *A. fatua, A. ludoviciana* and *A. sativa* are closely related, but taxonomically distinct, hexaploid species (2n=42). *A. barbata*, on the other hand, is a tetraploid species (2n=28) that is more distantly related to cultivated oats. Although it has long been recognized that the hexaploid species may be crossed readily to produce fertile hybrids, all these species are predominantly self-pollinated with natural intraspecific outcrossing rates typically less than 1%[2,5,8]. However, for an assessment of the potential consequences of gene flow between cultivated and wild oats, a measure of the actual extent of natural outcrossing between these species in field situations is needed.

Evidence for interspecific hybridization between A. fatua and A. sativa.
Interspecific outcrossing rates of 0.1% have been recorded[3] in artificial block plantings of *A. fatua* and *A. sativa*. We have extended this evidence to naturally occurring situations by assessing the occurrence of interspecific heterozygotes between *A. fatua* and *A. sativa* using electrophoretically detectable variants of marker loci specifying the enzymes alcohol dehydrogenase, esterase and leucine amino peptidase. In four mixed populations of *A. fatua* and *A. sativa* paired samples of the two species were collected and compared. In three of the populations (Populations 1 to 3; Table 1), *A. sativa* occurred as the occasional feral individual in almost pure *A. fatua* stands (roadside situations); in the fourth population, *A. fatua* was an occasional weed contaminant of a crop of cultivated oats.

Table 1. Estimates of outcrossing rates in four mixed populations of *Avena fatua* and *A. sativa* occurring in southern New South Wales.

Population	Species examined[a]	Number seeds tested	No. of hetero-zygotes (H)	Outcrossing rate (t)	S.E. (t)
1	A. sativa	362	3	0.83×10^{-2}	0.48×10^{-2}
2	A. sativa	107	1	0.93×10^{-2}	0.93×10^{-2}
3	A. sativa	80	0	0	—
4	A. fatua	220	1	0.45×10^{-2}	0.45×10^{-2}
Total		769	5	[b]0.65×10^{-2}	0.29×10^{-2}

a   Populations 1 to 3 dominated by *A. fatua*; population 4 dominated by *A. sativa*;
b   Weighted mean

In these natural stands the interspecific outcrossing rate varied between 0 and 0.93%, with a weighted mean of 0.69 ± 0.29% over all populations. These estimates fall within the general range of intraspecific outcrossing rates found for both species[2,5] and are consistent, although somewhat higher than, the interspecific estimates obtained by Derick[3]. However, they are especially important as they measure actual rates of outcrossing between the cultivated crop and its wild relatives in situations where these species typically occur together in mixed stands. In doing this they provide an estimate of the potential gene flow between these species in Australian environments.

Potential consequences of natural hybridization between A. fatua and A. sativa.
Continuing gene flow between the crop and weed species makes many of the problems that arise in attempts to control wild oats in cultivated oat crops more serious. For example, no herbicides are currently available which will discriminate between *A. fatua* and *A. sativa*. One proposal that has been advanced to solve this problem is to use the techniques of molecular biology to incorporate a gene for herbicide tolerance into *A. sativa*. However, the small but constant gene flow from *A. sativa* to *A. fatua* means that such a strategy would have a short effective life in the field. Attempts to discriminate chemically against *A. fatua* would simply produce a strong selection pressure favouring the rapid spread and assimilation of the tolerance gene into the weed species. This approach would also have significant implications for the control of wild oats in other cereal crops.

Problems of this nature do not occur with respect to the development of strategies to control infestations of *A. barbata* in cultivated oat crops. As *A. barbata* is incompatible with *A. sativa*, the two species are genetically isolated and no gene flow occurs. In these circumstances, incorporating a gene for herbicide tolerance into *A. sativa* would provide a long-term method of discriminating against *A. barbata* in mixed stands.

Whether such a strategy would be cost-effective would depend on an assessment of the relative importance of *A. barbata* as a weed of cultivated oat crops.

Wild oats and the epidemiology and micro-evolution of rust diseases

The role of wild oats in the off-season survival of *Puccinia coronata* and *P. graminis avenae*. All three species of wild oat in Australia act as alternative hosts for *P. coronata* and *P. graminis avenae*. As the sexual hosts of both these pathogens (*Rhamnus* spp. and *Berberis* spp., respectively) are virtually absent, both must rely on uredial infections to survive through the summer months. During this period, the incidence of cultivated oats is restricted to volunteer plants growing on roadsides. In contrast, wild oat plants are far more common occurring widely in response to localized favourable conditions.

As a consequence of a continuing long-term assessment of racial variation in *P. coronata* and *P. graminis avenae*, we have observed that wild oat plants are frequently infected with either or both rusts throughout the summer and into the following autumn. It is an inevitable conclusion that these wild species play an important role as offseason refuges for the pathogen populations attacking cultivated oats.

In addition, the explosion in wild oat population numbers that occurs in response to the first widespread autumn rains produces a flush of potentially susceptible host material well in advance of the oat crop sown in autumn. These plants provide a host resource for the early increase of both rust pathogens and hence a launching pad for the development of rust epidemics on newly planted crops.

Wild oat populations and micro-evolution of virulence in *P. coronata* and *P. graminis avenae*. There is considerable circumstantial evidence supporting the contention that wild oat populations are an important selective force favouring the appearance of new pathotypes of both *P. coronata* and *P. graminis avenae*.

At a gross comparative level, there is a marked difference in the number and diversity of pathotypes of *P. coronata* and *P. graminis avenae* occurring on oats and that of the ecologically equivalent pathogens found on wheat (*P. recondita tritici* and *P. graminis tritici*). Over the period 1980-1989, the average number of pathotypes detected and the diversity of the pathogen populations occurring in northern N.S.W. and Queensland were greater for both of the oat rust pathogens than their wheat rust pathogen equivalents (Table 2). This was the case despite: i) the total area of wheat grown in this region was substantially greater than that sown to cultivated oats; ii) the number of differentials used to distinguish pathotypes of the two wheat pathogens was greater than that for the oat rusts (number of differentials used for Prt, Pgt, Pc and Pga were 14, 16, 10 and 7 respectively); and iii) the number of different cultivars of wheat grown in the area was greater than the number of oat cultivars grown. These three differences should normally favour a greater range of pathotypes among the wheat pathogens. On the other hand, the most obvious difference in the epidemiology of the respective pathogens with which the patterns noted in Table 2 can be correlated, is the widespread and common occurrence of alternative hosts for the oat rusts and the relative absence of such hosts for the wheat rust pathogens along with the relatively high proportion of resistant genotypes.

For wild oat populations to exert strong selective pressure for a diversity of avirulence/virulence phenotypes in co-occurring pathogen populations they must, in turn, possess a diversity of resistance/susceptibility phenotypes. We found this type of variation to occur in eastern Australian populations of *A. barbata* and *A. fatua* with respect to resistance to both *P. coronata* and *P. graminis avenae*[1]. In that study, marked differences in the frequency of different seedling resistance infection types were apparent when 21 different populations of *A. barbata* and *A. fatua* were challenged with 8 different pathotypes of *P. coronata* and *P. graminis avenae*. Within individual populations, differences were observed in both the response of individual wild oat accessions to different pathotypes of the one pathogen, and between different accessions in their response to the same pathotype. Similarly, differences were detected in the response profiles of whole populations within a local epidemiological region and between populations in different regions. In the latter situation, populations of both *A. barbata* and *A. fatua* occurring in northern N.S.W. showed a higher overall level of resistance, and were more diverse in their response to pathotypes of either pathogen than were populations of these wild grasses from southern N.S.W.

Table 2. The number of isolates, number of different pathotypes and the diversity of populations of *P. coronata* and *P. graminis avenae* and their ecological homologues on wheat, *P. recondita* and *P. graminis tritici* detected on oats (wild and cultivated) and wheat, respectively, in northern N.S.W. and Queensland in the period 1980-1989 inclusive (excluding 1984-85 season for which data are incomplete).

	Oat rust pathogens		Wheat rust pathogens	
	P. coronata	*P. g. avenae*	*P. recondita*	*P. g. tritici*
No. of isolates	933	337	826	573
No. of pathotypes	51	16	26	20
Mean no. of pathotypes	15.7	7.6	7.0	5.4
E(H')[a]	2.896	2.063	1.913	1.523
± S.E.	0.002	0.002	0.002	0.002

a   Corrected Shannon-Weaver diversity index with standard errors[4].

The potential effect of this range of resistance phenotypes on populations of *P. coronata* and *P. graminis avenae* was investigated by comparing the number of pathotypes detected, the pathotypic diversity of populations and the avirulence/virulence phenotypes of rust populations collected during yearly routine rust surveys (1980 to 1989) from wild or cultivated oats in northern N.S.W., southern N.S.W. and Victoria (Table 3). For both pathogens, both the average diversity of the population and the average virulence of each isolate was least in Victoria and greatest in northern N.S.W. These trends are in agreement with those found previously in surveys of the southern and northern N.S.W. populations[7] and reflected the pattern of increasing variability and overall resistance in wild oat populations between southern and northern N.S.W.[1].

Comparison of the diversity and virulence of pathogen populations collected from wild and cultivated oats in the same geographic region found no significant differences. However, there was a consistent tendency for the average virulence of pathogen isolates collected from cultivated oats to be marginally higher than those collected from wild oats (Table 3). We believe this reflects the presence of a substantial number of plants in the wild oat population that are susceptible to even the most avirulent pathogens, while the majority of cultivated oat varieties are at least partially resistant to these races. This difference was most noticeable in *P. graminis avenae* where the occurrence of race 1 (carrying no virulence genes) is substantially higher on wild than cultivated oats ($\chi^2_1 = 12.39$; $P < 0.001$).

To date, breeding for rust resistance in *A. sativa* has not had a significant effect in Australia. As a consequence, little is known about the number and identity of the genes for resistance to *P. coronata* or *P. graminis avenae* that are present in Australian oat cultivars. Until this information is available, the extent to which variation for resistance in the cultivated oat crop contributes to the diversity of the overall *P. coronata* and *P. graminis avenae* populations will remain unclear. However, there can be little doubt that the very large and diverse populations of wild oats that occur in Australia are responsible for a substantial portion of the pathotypic variability that is observed.

Table 3. The number of isolates, the total number of pathotypes, the mean virulence of individual isolates and the pathotypic diversity of the total *P. coronata* and *P. graminis avenae* populations detected on wild and cultivated oats in Victoria, southern N.S.W. and northern N.S.W. over the period 1980 to 1989 inclusive (excluding 1984-85 season for which data are incomplete).

| | Region of origin of pathogen isolates: | | | | | |
| | Victoria | | Southern NSW | | Northern NSW | |
	WO[a]	CO	WO	CO	WO	CO
P. coronata						
No. of isolates	72	46	217	163	235	255
No. of pathotypes	14	12	32	28	32	35
Virulence[b]	2.89	3.28	3.35	3.97	4.23	4.49
E(H')[c]	1.75	1.84	2.32	2.45	2.65	2.71
± S.E.	0.02	0.03	0.01	0.01	0.01	0.01
P. graminis avenae						
No. of isolates	102	105	185	116	162	40
No. of pathotypes	10	12	12	14	13	10
Virulence	1.08	1.73	1.64	2.20	1.74	2.45
E(H')	1.56	1.79	1.80	1.92	1.97	1.85
± S.E.	0.01	0.01	0.004	0.01	0.004	0.02

[a] WO = wild oats; CO = cultivated oats; [b] mean virulence of isolates determined by averaging either the number of susceptible differentials (*P. coronata*) or the number of resistance genes (*P. graminis avenae*) across all isolates. [c]Corrected Shannon-Weaver diversity index with standard errors[4].

Conclusions

The close genetic and pathological relationship between *A. sativa* and the three species of wild oat found in Australia poses considerable problems for the health and hygiene of cultivated oat crops. In addition to acting as over-summering refuges for *P. coronata*, *P. graminis avenae* and potentially a range of other pathogenic organisms (for example, barley yellow dwarf virus), wild oats also favour the development and maintenance of a wide range of pathotypes of these pathogens.

The continuing outcrossing between *A. sativa* and its hexaploid wild oat relatives also has significant implications for both weed and disease control. Potential options in weed control are reduced by the likelihood that any gene for herbicide tolerance that is inserted into oat cultivars will move into at least two of the weedy species (*A. fatua, A. ludoviciana*) against which the strategy was originally conceived. In addition, however, this movement of genes from cultivated to wild species ensures that any novel resistances introduced into crop varieties from elsewhere will eventually flow through to the weedy species.

In turn, this may prove to be a serious obstacle to the successful use of a regional gene deployment strategy for disease control[6].

Acknowledgements

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Simulation of the Effects of Herbicide and Crop Rotation Practices on the Population Dynamics of Wild Oats

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Summary

A simplified simulation model of the wild oat life cycle was used to study the effect of varying the degree of kill or herbicide efficacy on long-term seed population trends in continuous wheat and wheat-fallow rotations. A herbicide efficacy of at least 85% would be required to prevent a rapid increase in the wild oat population in the continuous wheat rotation. A wheat-fallow or wheat-pasture rotation, which provides complete seed-set control in alternate years, enables the relaxation of herbicide selection pressure on wild oats in the crop as well as resulting in a rapid decline in the wild oat seed bank.

The Model

A weed seed bank operates in a similar way to a business enterprise. A simplified life-cycle model for wild oats includes the proportion of the seed bank (capital) which produces mature plants in the crop (investment), the number of seeds produced (return on investment) and the mortality of seeds in the seed bank (depreciation) (Figure 1). Wild oat plants surviving to maturity in the crop are assumed to represent five per cent of seeds present in the soil at the time of harvest of the previous crop.
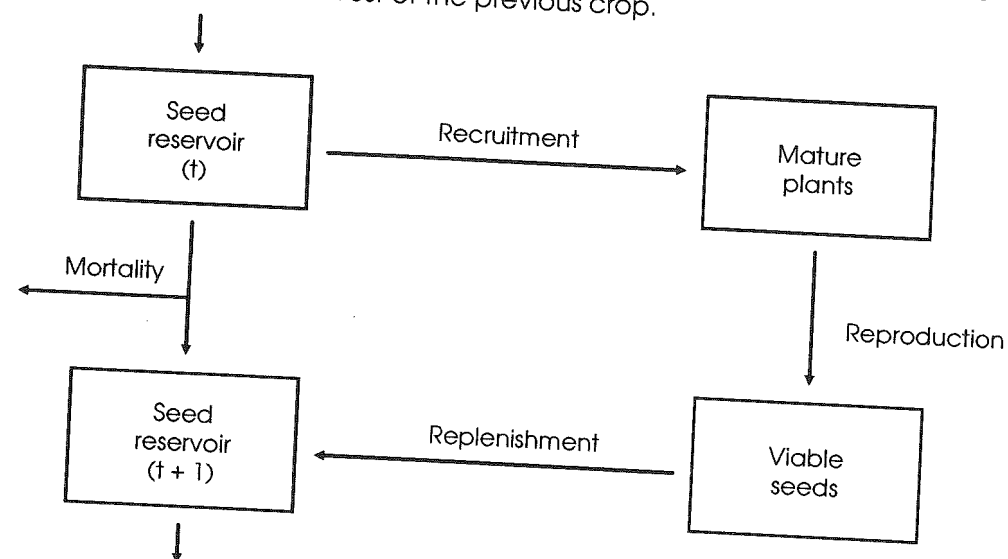

Figure 1. Simplified schematic representation of the wild oat life cycle.

The extent to which selective herbicides can prevent a build-up of wild oats under continuous cropping will depend on their efficacy or degree of kill. Pre-emergence soil-applied herbicides such as tri-allate are generally less efficacious than post-emergence herbicides such as diclofop-methyl for the control of wild oats.

The number of wild oat seeds produced is a function of the number of plants at maturity and approaches a limit of around 28 000 seeds per square metre in a crop with a yield potential of four t/ha (Figure 2). It is assumed that wild oat seed production would be proportionately less in crops with a lower yield potential.

Wild oat seeds exhibit greater levels of dormancy than other grass weeds such as *Bromus* and *Lolium* spp. However, if wild oats are prevented from seeding, the number of seeds in the soil declines at an exponential rate. A field study in northern NSW (Martin and Felton pers. comm.) has shown wild oat seeds to be relatively short-lived in the soil, with a half life of approximately six months (Figure 3).
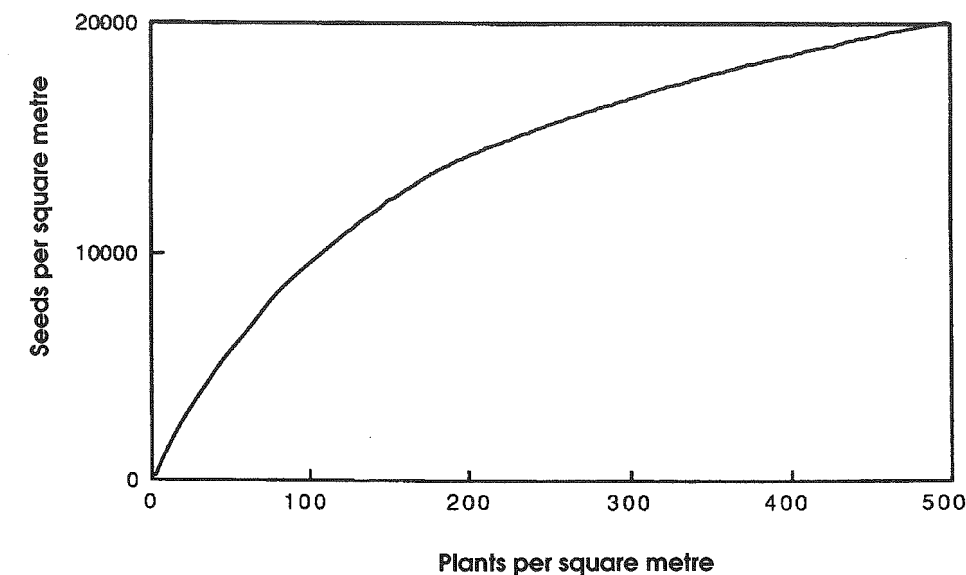

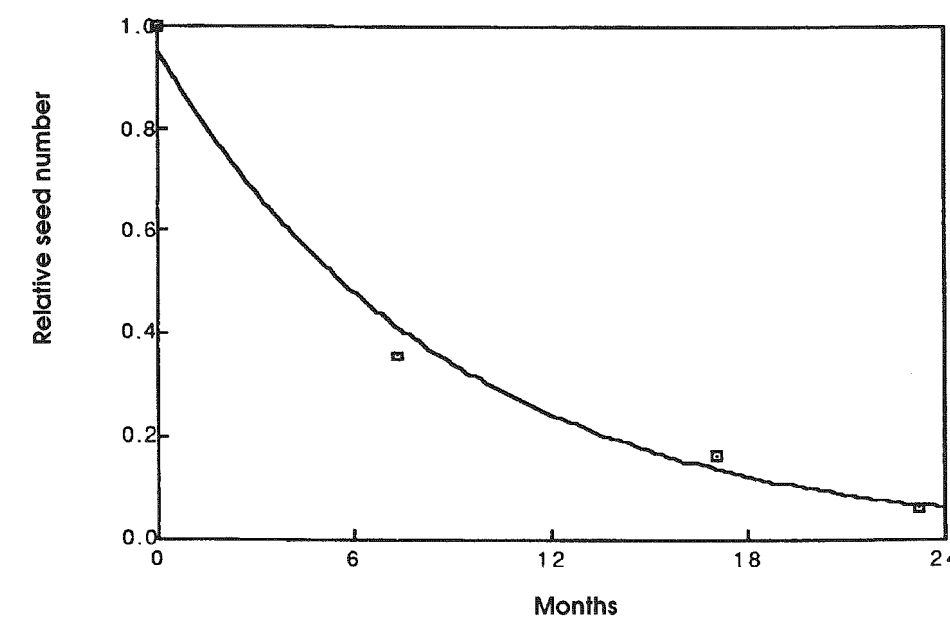**Figure 2.** Relationship between wild oat plant density and seed production.

Figure 3. Decline in the number of viable wild oat seeds in the soil over a period of two years.

Although a small proportion of seeds is removed at harvest, most losses are accounted for by seedling mortality during fallow weed control operations. A balance is eventually reached between seed production and seed mortality and for wild oats the maximum soil seed density possible is in the order of 15 000 seeds per square metre.

Results and Discussion

The effect of varying herbicide efficacy in continuous wheat and wheat-fallow rotations for 10 year periods is shown in Figure 4a and b. With a starting wild oat soil seed density of 100 seeds per m^2, a herbicide giving 75 per cent control permits a rapid increase in the seed bank (Figure 4a). In contrast, a herbicide giving 85 or 95 per cent control permits a marginal increase in the seed bank. Although a more efficacious herbicide will enable control of wild oats in a continuous wheat rotation, such a practice provides strong selection pressure for herbicide resistance in the wild oat population.

In a wheat-fallow or wheat-pasture rotation (Figure 4b), where wild oat seed-set is prevented in alternate years, use of a herbicide giving 75 per cent control in the wheat crop was sufficient to cause a progressive decline in the wild oat seed bank.
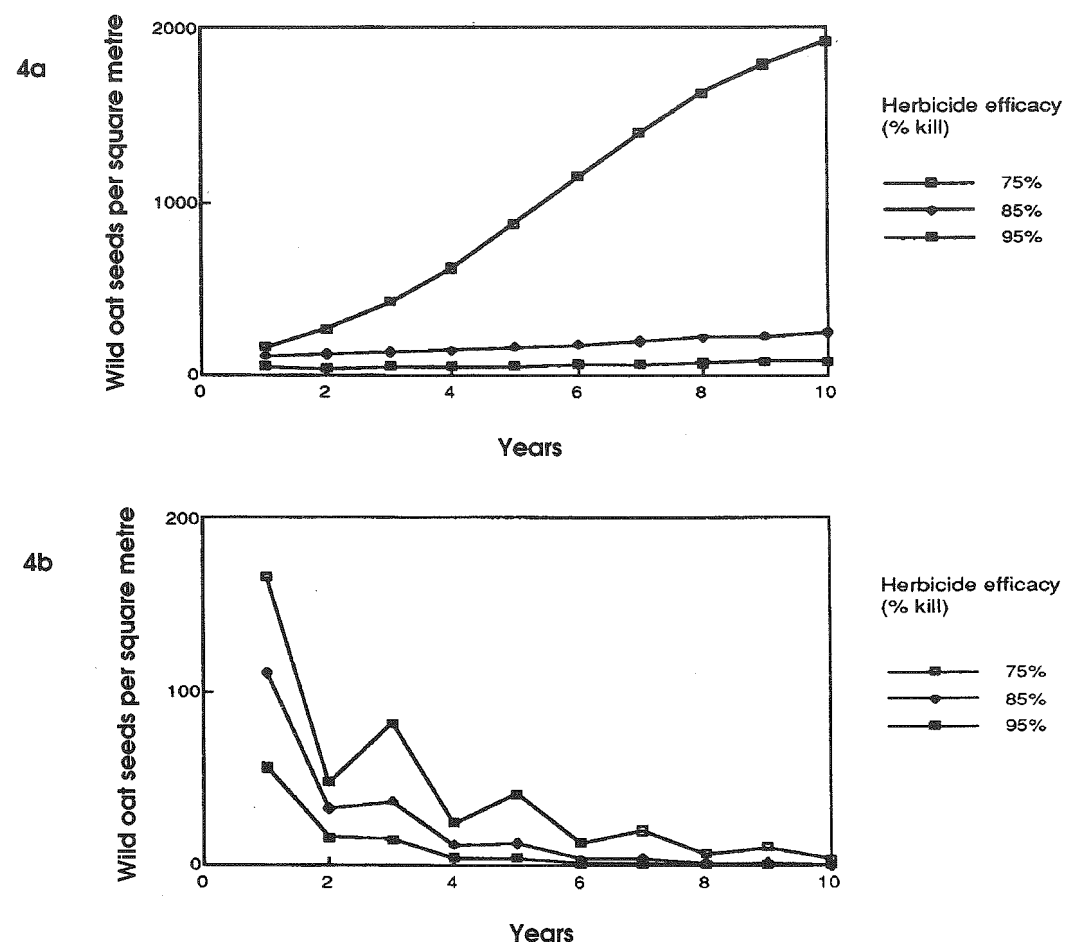
Figure 4. Effect of crop rotation and herbicide use on the wild oat seed bank.
(a) continuous wheat (b) wheat-fallow.

Simulation modelling of weed populations is therefore an invaluable tool for predicting the outcome of proposed changes to crop rotations, tillage practices and herbicide use on the farm.

Towards Eradication of Wild Oats in The Netherlands

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Summary

Because wild oats were rapidly declining in The Netherlands, an eradication program was begun in 1982 to rout a newly discovered infestation on the Statefarm in the IJsselmeerpolders region. After four years of treating infestations only with selective herbicides, new strategies consisting of a full inventory of infested fields, preventative measures to avert new infestations and curative measures to control known infestations had to be implemented with the objective of preventing seed shed. The program has cost around Hfl 4.8 million over the 10 years and has substantially reduced the area infested such that in the future only surveillance and hand roguing will be practiced for as long as is necessary to achieve eradication.

Introduction

Wild oats, predominantly *Avena fatua* L., were once among the most important weeds in The Netherlands. Changing agricultural practices, crop husbandry and the instigation of legislative measures under the country's 'Wild Oat Order' have brought about a decline in the importance of wild oats since the early 1960's[1]. The discovery in the early 1980's of a new infestation on a recently reclaimed Statefarm of the Ministry of Public Works in the IJsselmeerpolders region was thus viewed with alarm. Not only did this go against the declining trend, but the invasiveness of wild oats seriously threatened to devalue this prime broadacre agricultural land.

This contribution reports on a program initiated ten years ago to eradicate wild oats from the Statefarm, highlighting the strategies used, progress towards eradication and the cost involved.

Methods

The Statefarm originally consisted of approximately 20,000 ha divided into seven sub-farms, with symmetrically ordinated fields of 200 ha. Eradication of wild oats commenced in 1982. However, four years into the program after only using herbicides to treat infestations, changes became necessary since the weed was not being contained. New strategies designed to prevent seed shed were implemented in 1986. These consisted of a full inventory of infested fields, preventative measures to avert new infestations and curative measures to control known infestations.

Inventory

A team of 15 people was recruited to map infestations on each sub-farm during the cropping seasons between April and August. Scouts traverse fields annually, implanting markers on infestations. The markers are visible above the crop enabling surveyors with theodolites to ordinate infestations which in turn allows compilation of precise area by distribution maps and statistical details. Two years were required to complete the original inventory and the plan is for the inventory procedure to continue for as long as is required to achieve eradication of wild oats on the Statefarm.

Preventative measures

Two strict hygiene measures have been implemented. These consist of only using crop seed produced on fields free of wild oats and ensuring that machines used to work or harvest infested fields do not enter clean fields. As far as practicable, operations are organised so that designated machinery only works within fields categorised into light, medium or heavy infestations.

Canola is the second most important crop in the rotations practiced on the Statefarm. It presents a particular problem since no selective herbicides are registered in The Netherlands for control of wild oats in canola. Consequently, canola has been grown only on fields infested with wild oats in patches. These patches were destroyed, initially with either paraquat or glyphosate, but this method was discontinued because total prevention of seed set could not be achieved. Now, all herbage is removed from patches and buried and when necessary patches are cultivated to destroy wild oat seedlings which subsequently emerge.

Curative measures

Initially, infestations in cereals were controlled only by applying difenzoquat, the one selective herbicide registered for control of wild oats in The Netherlands. After 1986 additional procedures were implemented to achieve the stringent objective of preventing seed shed. Densely infested crops are destroyed using the methods outlined for patches in canola fields (see above); medium and lightly infested fields are sprayed with difenzoquat and hand rouged. Very light infestations are hand rouged once or twice each year after the panicles become visible above the crop.

Results and Discussion

Once wild oats has been eradicated from contiguous areas, land under Statefarm control is leased to private farmers. Due to this policy the Statefarm has been halved in area from approximately 19,000 ha to 9,500 ha within 10 years, and the area sprayed with difenzoquat reduced from a maximum of 30% in 1988 to 7.3% in 1991 (Figure 1). Because of the added difficulties in dealing with wild oats in canola crops, the majority of the infested area has been sown to spring barley, winter wheat or spring wheat (Figure 2). Although spring sown crops are less competitive than winter crops and favour wild oats, they have the advantage of allowing the weed to develop above the canopy, making observation and hand roguing more effective.

The proportion of the area treated with herbicides increased from 0.2% in 1982 to about 30% in 1988 (Figure 1). This trend emphasises that wild oats would have continued to spread with infestations becoming more dense, thus requiring even greater inputs of herbicide had the other measures not been implemented in 1986 to prevent seed shed. After two years under the policy of preventing seed shed the area of cereal crops sprayed has progressively declined (Table 1), apart from 1991 when additional use was made of difenzoquat as it was scheduled for deregistration in The Netherlands in 1992.

Table 1. Total area (ha) of cereal crops grown on the Statefarm between 1987 and 1991 and the proportion sprayed with difenzoquat.
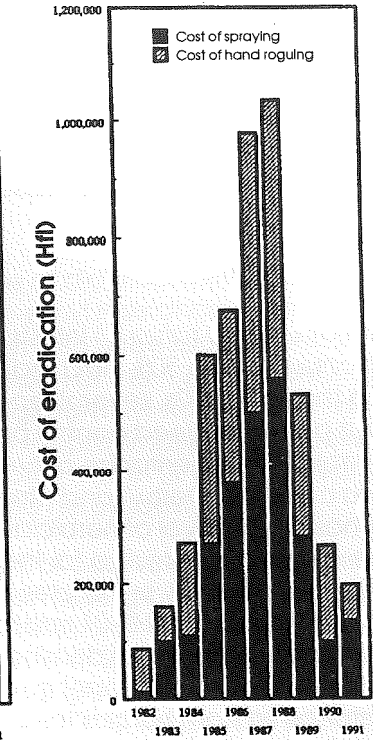
	1987	1988	1989	1990	1991
Total area of cereals (ha)	6630	6354	5798	4939	4458
Sprayed area in cereals (ha)	2322	2618	1503	529	693
Proportion sprayed (%)	35	41	26	11	16
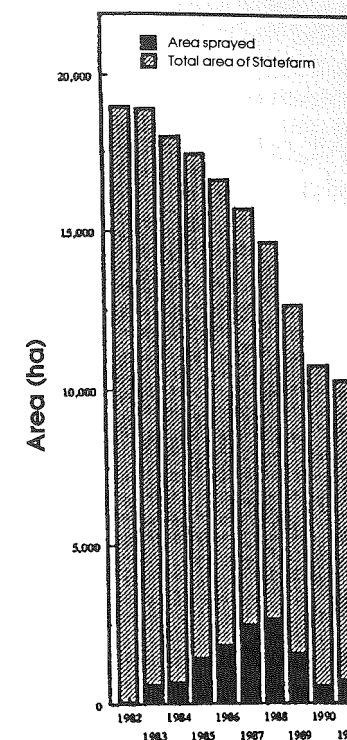
Figure 1. Total area of Statefarm and area sprayed with wild oat herbicide between 1982 and 1991
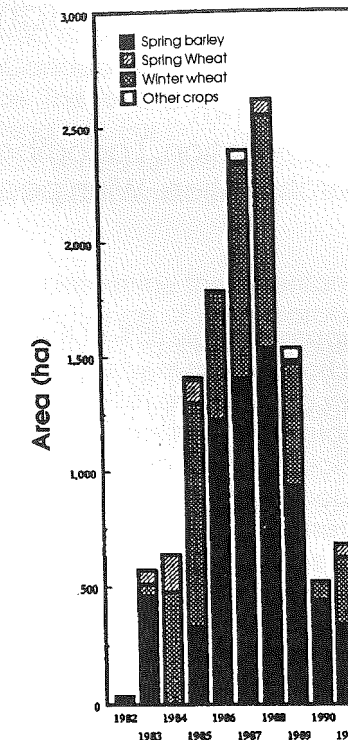
Figure 2. Crops grown on areas sprayed with wild oat herbicides between 1982 and 1991

Figure 3. Annual cost of wild oat eradication apportioned to hand roguing and herbicide control

Total cost of the program over the 10 years has been approx. Hfl 4.8 million. The annual cost peaked in 1987/88 at around Hfl 1.0 million and declined to approx. Hfl 0.2 million in 1991 (Figure 3). These costs to date have been about equally spent on treating infestations with herbicides and hand roguing (including surveying). In the future, only hand roguing will be carried out, and this will continue for as long as is necessary to achieve eradication.

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Genetic Studies on Herbicide Resistance in Oat

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Summary

Wild oat (Avena fatua L.) infests approximately 85% of the farms on the Canadian prairies, and causes a 5 to 50% yield reduction in fields in which it is the dominant weed species. Since the late 1980s, several reports of herbicide resistant wild oats have raised concern about potential weed control problems in Western Canada. This study was conducted to determine the inheritance of herbicide resistance in two A. sativa x A. fatua crosses. The A. sativa cultivars, 'Derby' and 'Random' were crossed with an A. fatua genotype, GP-HR-01, that has a high level of herbicide resistance. Parents, F_2s and $F_{2:3}$ families resulting from these crosses were tested for their reactions to two wild oat herbicides, diclofop-methyl and fenoxaprop-p-ethyl, in the greenhouse. The parents, cvs. Derby and Random, were susceptible, and GP-HR-01 resistant to both herbicides. Inheritance to diclofop-methyl and fenoxaprop-p-ethyl was dominant and monogenic in the Derby/GP-HR-01 cross, but was controlled by two dominant complementary genes in the Random/GP-HR-01 cross. Resistance to both herbicides appeared to be controlled by the same gene or groups of tightly linked genes.

Introduction

Wild oat (Avena fatua L.) infests approximately 85% of the farms in Alberta, Saskatchewan and Manitoba, and causes a 5 to 50% yield reduction in fields in which it is the dominant weed species. The economic loss to farmers as a result of these yield reductions, and the cost of herbicides used to control wild oat in Canada is estimated at over $280 million.

Over the last 15 years, several germplasm lines have been identified by geneticists and plant breeders as potential sources of herbicide resistance, and some of these germplasm lines have been utilized in oat breeding programs. Savena I, cultivar resistant to diclofop-methyl, was developed and registered in Australia[1] and several other germplasm lines have been released in the United Kingdom[3]. Brown and McKenzie[2] and Warkentin et al. [4] have reported that two independent recessive genes controlled diclofop-methyl resistance in Savena I and related germplasm, and suggested that breeding for herbicide resistance should be relatively easy.

In 1990, a herbicide resistant A. fatua genotype, hereafter referred to as GP-HR-01 was discovered in a farmer's field in which wild-oat herbicides were used over a long period of time. Greenhouse evaluation of GP-HR-01 later revealed that this A. fatua genotype can tolerate over ten times the commercially recommended rates of diclofop-methyl and fenoxaprop-p-ethyl application (Leoppky pers. comm.).

The main objective of this study was to determine the types of gene action, and the number of genes conditioning herbicide resistance in two A. sativa x A. fatua crosses involving GP-HR-01.

Materials and Methods

GP-HR-01 is a diclofop-methyl and fenoxaprop-p-ethyl resistant genotype of A. fatua. Derby and Random are standard oat (A. sativa) cultivars registered for Western Canada. Both cultivars are sensitive to all known wild oat herbicides. GP-HR-01 was crossed with 'Derby' and 'Random' during the winter of 1991.

The F_1 plants from the Derby/GP-HR-01 and Random/GP-HR-01 crosses were grown in a growth chamber and self-pollinated to produce the F_2 seed. The F_2 seedlings were grown in a greenhouse at the Agriculture Canada Research Station in Lacombe, Alberta, and evaluated for herbicide resistance. The testing was done by growing the seedlings in plastic pots (15 cm diam.) filled with potting mix. For both crosses, six kernels per pot were planted approximately two cm deep, and thinned to five seedlings per pot. Herbicide evaluations were conducted in two separate experiments hereafter referred to as Experiment One and Experiment Two.

Experiment One was carried out to determine the inheritance of diclofop-methyl resistance, and Experiment Two studied the inheritance of fenoxaprop-p-ethyl resistance. Each experiment was arranged in a completely randomized design with three replications. In both experiments, a replication consisted of 10 seedlings of each of the three parents, 100 F_2 seedlings from the Derby/GPHR-01 cross, and 50 F_2 seedlings from the Random/GP-HR-01 cross. When the seedlings were in the two to three leaf stage (approximately two weeks after emergence), they were transferred to an automated spray booth (R & D Sprayers Inc. Opelousas, Louisiana 70570) and sprayed with the appropriate herbicides.

The parents and the F_2 seedlings grown in replications 1 and 2 of each experiment were sprayed with two times (2x) the recommended rates, whereas those grown in replication three were sprayed with three times (3x) the recommended rates of the herbicides. The herbicides were applied in 100 L ha^{-1} of water and at a pressure of 275 kPa using TeeJet® flat-fan nozzles. After spraying, the seedlings were placed in the greenhouse, and two to three weeks later were classified as either susceptible or resistant. The susceptible seedlings were withered and had severe necrosis, whereas the resistant seedlings were healthy and showed no lasting visible symptoms of herbicide injury. To verify the results from the F_2 data, additional experiments were carried out using a minimum of 60 $F_{2:3}$ families/cross, with each family consisting of at least 20 seedlings.

Results and Discussion

The reactions of the parents and the F_2 seedlings from the two experiments are presented in Table 1. All of the Derby and Random seedlings were completely killed with diclofop-methyl and fenoxaprop-p-ethyl, whereas the GP-HR-01 seedlings survived the 2x and 3x treatments of both herbicides.

In the Derby/GP-HR-01 cross the number of F_2 seedlings in the resistant and susceptible classes for each of the two herbicides fit the 3:1 ratio expected for monogenic inheritance. In the Random/GP-HR01 cross, the number of F_2 plants in the resistant and susceptible classes fit the 9:7 ratio expected for dihybrid inheritance (Table 1).

Table 1. The reactions of F_2 seedlings and $F_{2:3}$ families from two A. sativa x A. fatua crosses to two wild oat herbicides.

Cross	Herbicide	Expected F_2 ratio	χ^2	P	Expected $F_{2:3}$ ratio	χ^2	P
		(R:S)[1]			(R:Sg:S)[1]		
Derby/GP-HR-01	diclofop-methyl	3:1	0.592	0.50-0.25	1:2:1	2.429	0.50-0.25
	fenoxaprop-p-ethyl	3:1	0.143	0.75-0.50	1:2:1	1.042	0.75-0.50
Random/GP-HR-01	diclofop-methyl	9:7	0.734	0.50-0.25	1:8:7	0.163	0.95-0.90
	fenoxaprop-p-ethyl	9:7	0.482	0.50-0.25	1:8:7	0.597	0.75-0.50

[1] R= Resistant; Sg = Segregating and S = Susceptible.

In the Random/GP-HR-01 cross, the number of $F_{2:3}$ families that were homozygous resistant, segregating, and homozygous susceptible to each of the two herbicides gave a good fit to the 1:2:1 ratio expected if one gene conditioned resistance to diclofop-methyl and fenoxaprop-p-ethyl. In the Random/GP-HR-O1 cross, the number of $F_{2:3}$ families that were homozygous resistant, segregating, and homozygous susceptible to each of the two herbicides gave a good fit to the 1:8:7 ratio expected for two dominant complementary genes (Table 1). These results supported the F_2 data and confirmed that resistance to both herbicides was controlled by a single dominant gene in the Derby/GP-HR-01 cross, and by two complementary dominant genes in the Random/GP-HR-01 cross. Additional studies that we have completed more recently (data not presented here) indicated that the same set of genes that condition resistance to diclofop-methyl also confer resistance to fenoxaprop-p-ethyl.

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Studying Water Status and Phytochrome Action in Seed Germination of Wild Oat (*Avena fatua*), with Application of Nuclear Magnetic Imaging

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Summary

Interaction of water status and phytochrome action on germination of wild oat (*Avena fatua*) was studied using dormant seeds subjected to mechanical injury or after-ripened in different relative humidities. Deviant phytochrome action was observed in mechanically-injured seeds, i.e. brief red light inhibited germination which could be reversed by far-red light. Effects of brief red light on germination varied in seeds after-ripened in different relative humidities. Seed water status may play a determinant role in the process of photoreaction. Nuclear magnetic resonance imaging can reveal detailed water distribution at the organ level in germinating seed.

Introduction

The importance of water status in wild oat (*Avena fatua*), with respect to dormancy and germination, was demonstrated indirectly by mechanical injury to the seed coat, i.e. the pericarp plus the testa[9,10,11]. Piercing the seed coat substantially increased germination of dormant seed in certain genetic lines. This mechanical injury allowed easy access of water to the embryo and uptake of an extra amount of water essential to initiate germination[9]. Furthermore, the location of the injury could determine the expansion order of the radicle and the scutellum[10]. The response pattern of the organs depended upon which was closer to the site of injury. Normally the radicle expands prior to the scutellum in an intact germinating seed. These observations indicate that the amount of water and its distribution are separate aspects of the water status in a seed that influence germination behaviour.

Water status was also found to interact with light effects on germination of wild oat seed. Hsiao and Simpson[5] showed that red light promoted germination of seeds imbibed in relatively high volumes of water, but inhibited germination of those in low volumes of water. A recent study with a non-dormant line of wild oat demonstrated that prolonged far-red (FR) light inhibited germination of intact seeds in water[4]. Germination of the halved seeds, however, was not inhibited by the same light treatment unless they were imbibed in PEG solutions. When seeds from six dormant lines were tested with alternate brief red (R) and FR light at various after-ripening stages, they showed typical phytochrome-mediated responses to the light through a time period starting when their primary dormancy was just about lost (Hou and Simpson, unpublished data). In seeds which had been overly after-ripened, germination was equally high in water regardless of light treatments. Phytochrome action affected germination of those seeds only when they were imbibed in media of specific negative water potentials. The interactions of water status and light on the germination indicate that the photoreceptor may function by regulating water uptake by the seed.

The amount of water in a seed is measured traditionally by weighing the seed before and after it is oven-dried. This method, however, has limitations for measuring the distribution of water in various parts of a seed. It is an invasive approach that limits meaningful analysis of the relationship between water status and some other physiological processes that lead to germination. In this regard, proton (^1H) nuclear magnetic resonance (NMR) techniques provide promising means to non-invasively study water status in plant tissues[6]. There is a direct relationship between the spin-lattice relaxation time (T_1) of water protons and the water content in plant materials[7,8]. NMR imaging has been used to study water relations in

plant material as small as a developing wheat grain[6,2]. This latter approach may allow continuous observations of water distribution, water movement, and changes in water content during the process of seed germination.

Part of this study was to characterise interactions of water status and phytochrome action in dormant wild oat seeds during the early stage of after-ripening. Included were effects of alternate brief R and FR light on mechanically-injured dormant seeds, and effects of brief R light on seeds after-ripening in different RH. In addition, the application of NMR imaging to studying water status in wild oat seed during imbibition was explored.

Materials and Methods

Mechanical injury to seed coat and phytochrome action

Wild oat plants were grown in a controlled-environment chamber (day/night (16/8 h) temperature 20/16°C, RH 65/85%). Seed collection and storage were described earlier[4]. Freshly-harvested secondary seeds were dehulled by hand and used in the experiment. Mechanical injury was done with a surgical blade by slightly slashing the coat along the embryo line on one side of the seed. Alternate R and FR light (10 min each) was applied after 6 h of incubation in darkness at 20 ± 1°C. Details of the germination test, its assessment, and the light qualities have been described earlier[4] (Light source No. 1 for R, No. 4 for FR in Table 1). Experiments were repeated at least twice.

After-ripening in different RH

Plants of a dormant line (CS166) were grown in a greenhouse. Dehulled primary seeds were dusted with Captan fungicide and placed in sealed desiccators with 0, 30, 60, and 90 percent RH respectively. The RHs were monitored with $CaCl_2$ granules or solutions according to(13). The desiccators were kept in darkness at 20 ± 1°C. Germination tests were carried out after 3, 6, 9, 12, and 15 weeks after-ripening (10 seeds per Petri dish, either treated with 10 min R or in darkness). The experiment was repeated twice.

NMR imaging

The instrument used was a Bruker 200 MHz Biospec unit (4.2 T). A glass tube (3.5 mm internal I.D., 5 cm long) was used to facilitate the positioning of the seed in the radio-frequency (r.f.) coil. One end of the tube was tapered and broken at the tip to form a tiny hole for air exchange. A seed, previously imbibed in water for a fixed time and blotted dry briefly on tissue paper, was inserted into the tube from the opposite end and fixed in place with a piece of plastic net. Moist cotton was placed a few millimetres away from the seed. A four-turn receiver coil (5 mm I.D.) was superimposed on the sample tube to achieve better signal-to-noise ratio. A Bruker multislice variable echo pulse sequence was used to generate images. The imaging gradients, timings of pulse intervals, as well as number of accumulations were varied to obtain the best possible images, but time spent for accumulating an image was usually about 30 min or less. Images were recorded as 256 x 256 pixel arrays.

Results and Discussion

Unusual responses to the alternate R and FR light treatments were observed from the mechanically-injured dormant seeds. Germination after a brief terminal FR exposure was higher than that after single or terminal R. The differences were especially significant during the early period of incubation. For example, in one line (AN265) after two days incubation, germination in darkness was 50%, indicating removal of the coat-imposed dormancy in part of the seed population[12]. Germination after exposure to R or R-FR-R was about 20%, while after R-FR it exceeded 60%. After 14 days incubation, germination in darkness, after R, R-FR-R, and R-FR was 60, 40, 60, and 70% respectively. Similar responses were observed in other dormant lines such as AN87 and SH99.

The relative water contents in CS166 seeds after-ripening for 15 weeks in 0, 30, 60, and 90% RH were 4.05 ± 0.61. 9.11 ± 0.65, 15.09 ± 0.68, and 24.86 ± 1.06% respectively. Germination in darkness of the seeds after-ripening in 0 and 90% RH was much lower than those in 30 and 60% RH throughout the testing period. This indicates a slow progress of terminating primary dormancy when seeds were in the two extreme moisture conditions. Brief R light was always inhibitory to germination of the seeds after-ripening in 0% RH. This R light significantly promoted germination of seeds in 30 or 60% RH during their later stages of after-ripening. Effects of the R light on germination of seeds in 90% RH were not significant. Germination of R-treated seeds changed, with progress of after-ripening, from lower to higher than dark germination.

The above responses to light were apparently mediated by phytochrome since they were R/FR reversible[1]. Inhibition of germination by the active form of phytochrome (P_{fr}) was reported in a dark-germinating species, Bromus sterilis [3]. In the same lines of wild oat tested in this study, however, the action of P_{fr} was proved to be normal, i.e. promoting germination in intact seeds when primary dormancy was about to be terminated (Hou and Simpson, unpublished data). The deviant phytochrome action was obviously caused by the differences in seed water status. Thus water status may play a decisive role in the process of photoreaction that includes interception of light signals by the photoreceptor, conduction of the signals, and final expression of the impact of light. For germination to occur, water status and phytochrome action must be in synchronisation with states of other components of the seed[12].

The NMR images allow detailed observation of water distribution at organ level such as the radicle, coleoptile, and scutellum, but not necessarily at tissue levels such as the aleurone layer. Some interesting features were revealed by the images. For example, in a germinating seed at an early stage of imbibition, a concentration of water was shown (by two orientations) along the interface between the scutellum and coleoptile. This was not expected since in an intact seed the most active organ was assumed to be the radicle that always expands first[10]. The observation needs further verification. On the practical side, satisfactory images can be acquired with a total accumulation time as short as 10 min. This time scale, plus carefully controlled air and moisture conditions in the sample tube, may allow use of the technique for further studies of interactions of water status with other environmental factors on germination without causing severe interruption of metabolic processes.

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Optimising Herbicide Use for Control of Wild Oats in Winter Crops

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Abstract

Herbicides are necessary to minimise or prevent yield loss due to competition from wild oats and to contain the spread of weed infestations. Reduced herbicide doses are desirable for economic and environmental reasons. Studies are in progress to determine ways to reduce or replace the use of herbicides for control of wild oats by examining the interaction between herbicide dose, crop competitiveness and environmental factors. Improved competitive ability may be achieved by choosing more competitive species or cultivars and also by manipulating crop density. The competitiveness of different crops and a large number of wheat cultivars are being examined to determine the range of competitive ability available to us. We are also examining the characteristics conferring competitiveness. In addition, because weed control by herbicides is influenced by environmental factors, herbicide doses may be reduced when optimal conditions occur before, at or after spraying. We are examining the influences of soil moisture, temperature and light on the performance of post-emergence wild oat herbicides to predict variation in dose-responses under different conditions, and also to determine the mechanisms underlying these responses. These results will provide additional strategies for future integrated control of wild oats in winter crops.